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Presenile Dementia with Alzheimer-, Pick- and Lewy-Body Changes

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Summary. An autopsy case of unclassifiable presenile dementia is reported. The outstanding pathological findings were as follows;

1. presence of senile plaques, neurofibrillary changes, Pick bodies, Hirano bodies, granulovacuolar degeneration of neurons, etc.
2. numerous Lewy bodies in the brain stem and diencephalon,
3. peculiar swollen neurons with intracytoplasmic, eosinophilic and argentophilic inclusions ("Lewy-like-bodies") in the cerebral cortices.

Detailed study of the last mentioned inclusions indicates that they are almost identical to Lewy bodies, though there are some minor differences, in histochemical and electronmicroscopic findings.

Nosologically, this case may represent either a combination of Alzheimer's disease, Pick's disease and idiopathic Parkinsonism with "Lewy-like-bodies" in the cerebral cortices, or a single disease.

As far as we know, no similar case been reported.

Key words: Lewy body — Idiopathic Parkinsonism — Alzheimer's disease — Pick's disease — Unclassifiable presenile dementia.

INTRODUCTION

Increasing numbers of cases with presenile dementia unclassifiable by the classical clinico-pathological criteria have been documented in Japan for the last 10 years. Kosaka et al. (1973) and Oyanagi et al. (1975) recently reported cases similar to those which McMenemy (1963) and Tariska (1970) called "Alzheimer's and Pick's double disease" or "superimposed Pick's and Alzheimer's disease". These cases were characterized by the simultaneous occurrence of many types of "senile changes" in the brain, in addition to the findings which characterize Pick's disease.

This report will deal with a case of presenile dementia, the neuropathology of which revealed numerous "Lewy-like-bodies" in the nerve cells of the cerebral cortices as well as typical Lewy bodies in the brain stem and diencephalon, in addition to such common "senile changes" as are found in the brains of Alzheimer's and Pick's diseases.

Thus, the present case may represent either a combination of Alzheimer's disease, Pick's disease and idiopathic Parkinsonism with "Lewy-like-bodies" in the cerebral cortices, or a single disease entity.

To our knowledge, no such case has been reported so far.

CASE REPORT

The patient was a 65 year old female. Family history disclosed no remarkable degenerative or hereditary illness. Her past history revealed rheumatism, diabetes mellitus, pyelonephritis and pulmonary tuberculosis in the climacterium.

At the age of 56, mild involuntary movement of the neck and forgetfulness were noticed by her daughter. Memory disturbance progressed gradually. She was hospitalized because of progressive dementia and psychomotor restlessness at the age of 65. Initial physical and psychic examination disclosed general emaciation, hypertension (180/120 mmHg), muscular rigidity of extremities, accentuated tendon reflexes, abulia and severe dementia. She was suspected of senile dementia with cerebral arteriosclerosis or Parkinsonism-Dementia Complex. She was restless and negativistic, but soon became unable to walk. Four months after admission, she suddenly died of intestinal invagination. Total duration of her illness was about 9 years.

Laboratory examination including E.C.G., routine blood tests and urinalysis, skull and chest radiographs revealed no noticeable abnormality. E.E.G. showed diffuse alpha activity.

Post-mortem examination revealed intestinal invagination, old pleuritis and contracted kidney.

Neuropathological Findings

Gross. The brain weighed 1130 g after 10% formalin fixation. There were diffuse cerebral atrophy and a mild degree of arteriosclerosis in the basilar arteries. On coronal sections

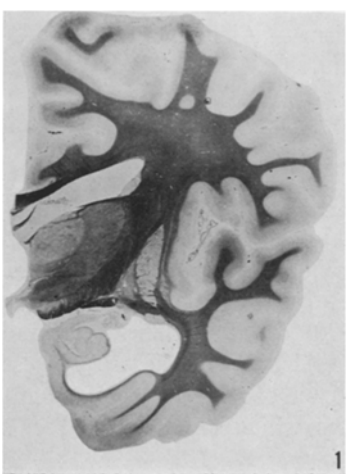


Fig. 1. Frontal section through the level of the thalamus and substantia nigra showing the localized atrophy of the left Ammon's horn, hippocampal gyrus and fusiform gyrus with dilatation of the inferior horn of the left lateral ventricle. (Luxol fast blue)

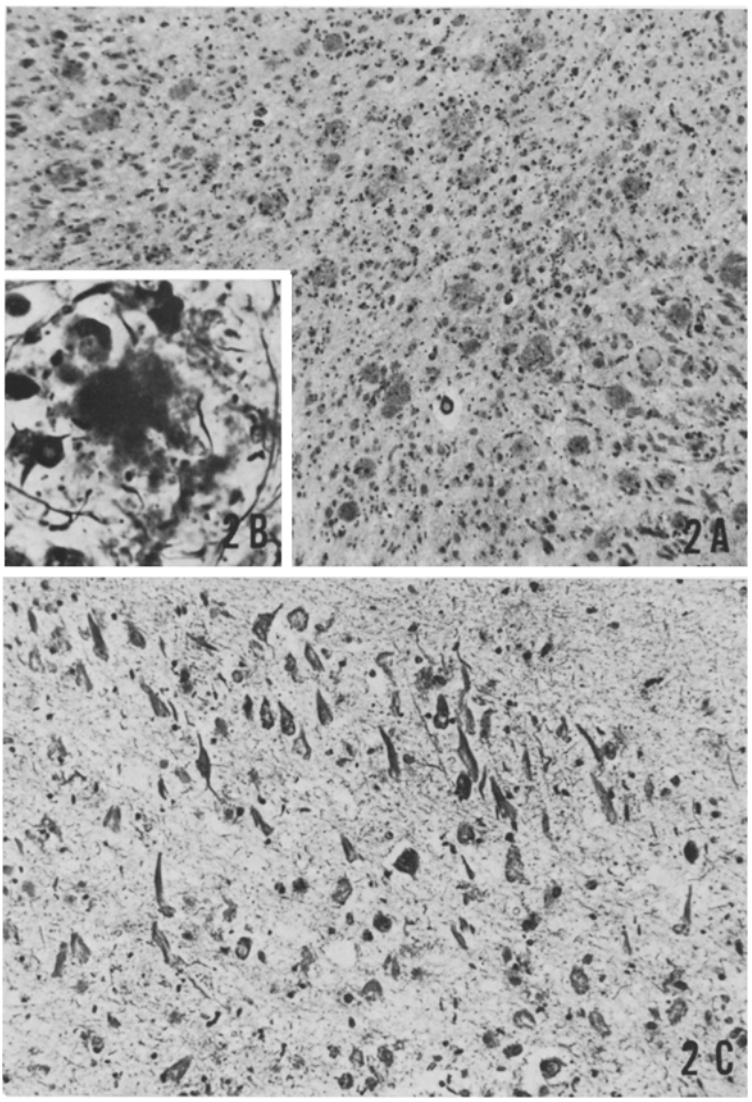


Fig. 2A–C. "Senile changes" in C.N.S. (A) Senile plaques in the hippocampus. (P.A.S., ×68). (B) Senile plaque in higher magnification. (Bodian, ×680). (C) Neurofibrillary tangles in the hippocampus. (Bodian, ×170)

of the brain, atrophy was much more accentuated in the Ammon's horns, hippocampal gyri and fusiform gyri, predominating in the left (Fig. 1).

Ligh Microscopy. Three groups of outstanding findings were identified. The first was the presence of various types of "senile changes", extensively distributed in the C.N.S. Senile plaques were diffusely distributed in the cerebral cortices (Fig. 2A and B). A few senile-plaque-like structures were also seen in the molecular layer of the cerebellum. Neurofibrillary tangles were found not only in the cerebral cortices, especially in the hippocampus (Fig. 2C), but also, though less in number, in the hypothalamus, substantia nigra

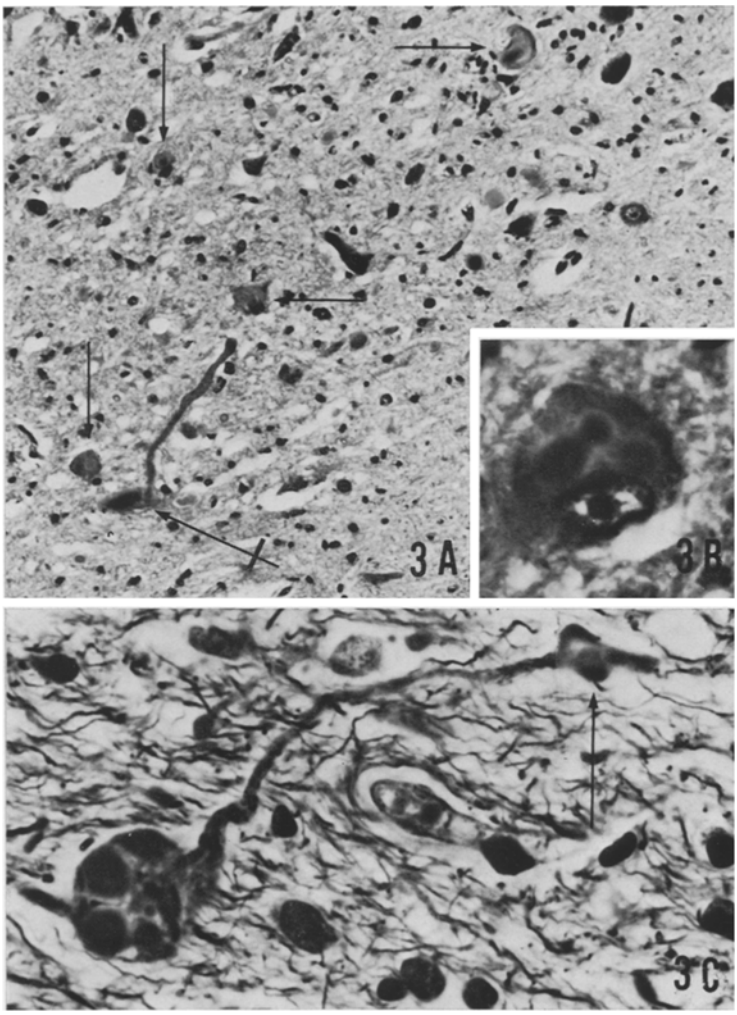


Fig. 3A—C. Lewy bodies in the diencephalon and brain stem. (A) Lewy bodies of typical type (arrow) and of cylindrical type (crossed arrow) in the dorsal efferent nucleus of the vagus. (H.-E., ×170). (B) Three Lewy bodies in a hypertrophied and degenerated neuron in the substantia innominata. (H.-E., ×680). (C) Four Lewy bodies in a neuronal perikaryon and one in the dystrophied axon (arrow) in the hypothalamus. (Bodian, ×680)

and superior central tegmental nucleus. There were abundant granulovaculoar degenerations and Hirano bodies in the Ammon's horn. In addition, numerous inflated cells with intracytoplasmic argentophilic bodies of Pick type were found in the amygdaloid nucleus, hippocampus and temporal cortex. Almost all neurons contained excess lipofuscin granules. There were also many torpedos in the cerebellar granular layers and spheroid bodies in the fasciculus gracilis.

The second outstanding finding was the presence of Lewy bodies in the substantia nigra, hypothalamic nuclei, substantia innominata, locus coeruleus, dorsal motor vagal nucleus (Fig. 3A—C). Most of them were typical ones having strongly eosinophilic cores

and pale halos in H.-E. staining. Occasionally, three or four Lewy bodies were found in a single neuron (Fig. 3B and C) and cylindrical forms of them were also detected. Distribution pattern of Lewy bodies in the brain of this case was almost identical to that of idiopathic Parkinsonism.

The third finding was the most characteristic one. Peculiar swollen cells with intracytoplasmic, eosinophilic and argentophilic inclusions were scattered widely in the cerebral cortices (Fig. 4A–H). Usually, a single, round or oval inclusion body was found in a single nerve cell. This type of neuronal degeneration were found mainly in small or medium sized pyramidal neurons in the deep layers in all cerebral cortices. In H.-E. preparations, these bodies appeared homogeneously eosinophilic, but less so than typical Lewy bodies. Almost all of them were ill-defined and without halos (Fig. 4B–E). In Bodian preparations, they were argentophilic, and fibrillary and granular in structure (Fig. 4F–H). Histochemistry revealed that these bodies were PAS negative, noncongophilic and non-sudanophilic. Millon, Sakaguchi, mercuric phenol blue and coupled tetrazonium reactions were all positive. They stained blue with Heidenhain azan, faintly blue with Kluever-Barrera and faintly brown with phosphotungstic acid haematoxylin staining. These histochemical properties were almost identical to those of Lewy bodies in the brain stem and diencephalon.

The neurons containing such bodies were swollen and showed nuclear eccentricity with loss or margination of Nissl substance. Occasional lipofuscin granules and rare granulovacuolar degenerations were found adjacent to the inclusions. Cellular reactions (e.g. neuronophagia, satellitosis) were not seen. The bodies were never observed in neuroglia or freely in the tissue.

Other pathological findings were as follows;

1. nerve-cell-loss and spongy state in mild degree in the second and third cortical layers of the rectal and hippocampal gyri,
2. fibrous gliosis in the hippocampal white matter, pallidum, substantia nigra and inferior olivary nucleus,
3. nerve-cell-loss and demyelination in mild degree in the pallidum,
4. nerve-cell-loss and depigmentation of melanin in moderate degree in the substantia nigra,
5. "grumose degeneration" in the dentate nucleus,
6. hyaline degeneration of cerebral arterioles.

Electron Microscopy. Small pieces of formalin fixed tissue from the Ammon's horn and the middle temporal gyrus were refixed in chrom-buffered 2% osmium tetroxide solution, embedded in Araldite resin and examined with a JEOL 100B electron microscope.

In 1 μ plastic sections stained with toluidine blue, many neuronal inclusions comparable to both Pick bodies and "Lewy-like-bodies" in the routine preparations were observed. In electron microscopy of corresponding areas, two types of inclusions were again observed in the cytoplasm of neurons.

The first type of them was a conglomerate of proliferated neurofilaments, 100 Å in diameter and with tiny side arms, intermingled with numerous mitochondria, a few cytoplasmic vesicles and ribosomes. The inclusions occupied a greater part of the cytoplasm displacing the nucleus. Other cytoplasmic organells were nearly absent (Fig. 5).

The second type of inclusions was composed of tightly packed filaments and granular materials (Fig. 6A). The filaments were crisscrossed randomly. In the central area, they were more compactly packed than in the peripheral zone of the inclusion (Fig. 6B), giving an appearance of a central electron dense core under low magnification. Individual filaments were not twisted as were seen in neurofibrillary tangles. Those in the central zone measured approximately 120–130 Å

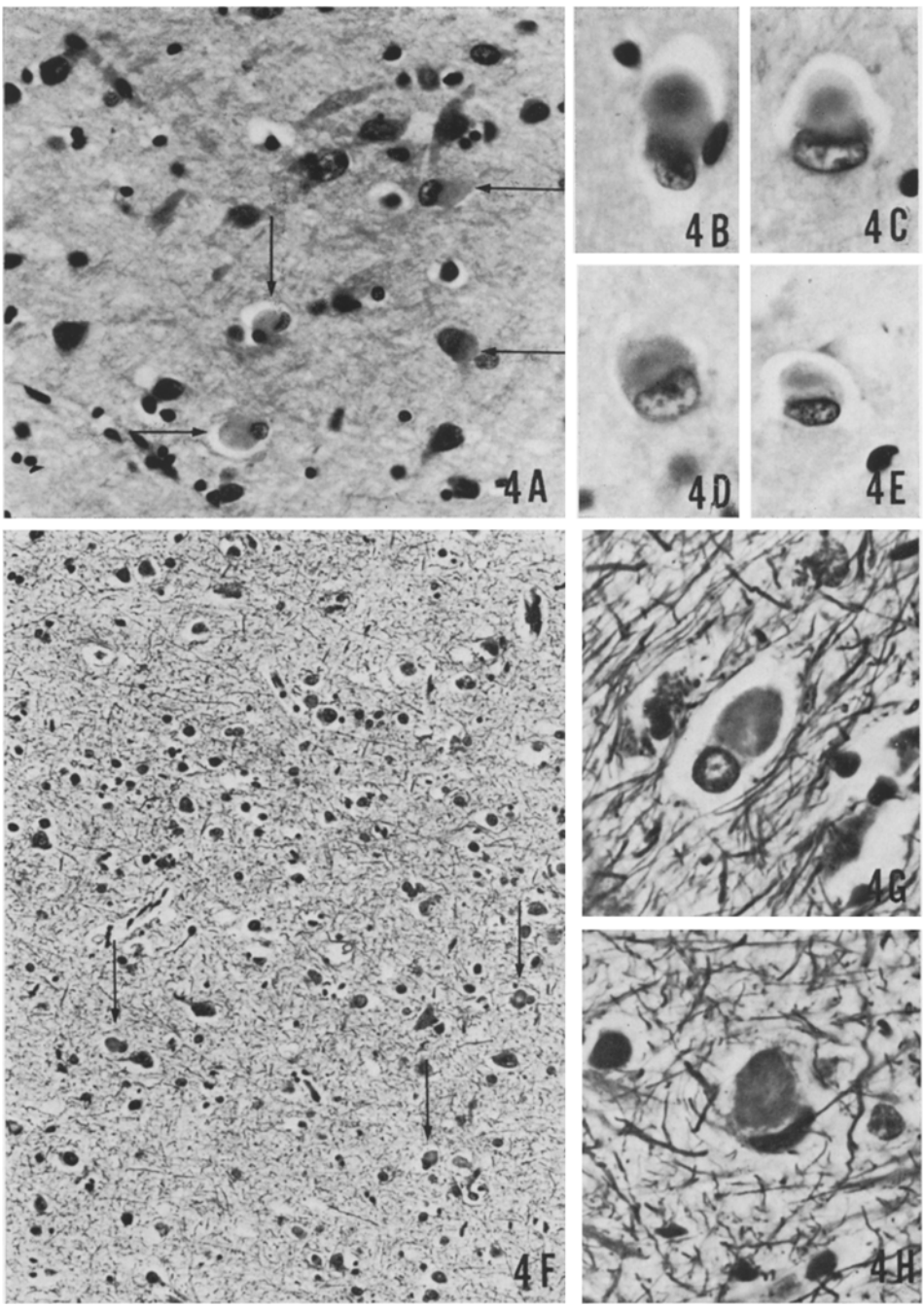


Fig. 4A—H. "Lewy-like-bodies" in the cerebral cortices. (A) Four peculiar swollen cells with "Lewy-like-bodies" and eccentric nuclei (arrow) in the deep layers of the insular cortex. (H.-E., ×170). (B—E) "Lewy-like-bodies" in higher magnification of H.-E. preparations. Their eosinophilia is variable in intensity. (×680). (F) Three peculiar swollen cells with argentophilic "Lewy-like-bodies" (arrow) in the deep layers of the temporal cortex. (Bodian, ×170). (G—H) "Lewy-like-bodies" in higher magnification of Bodian preparations. (×680)

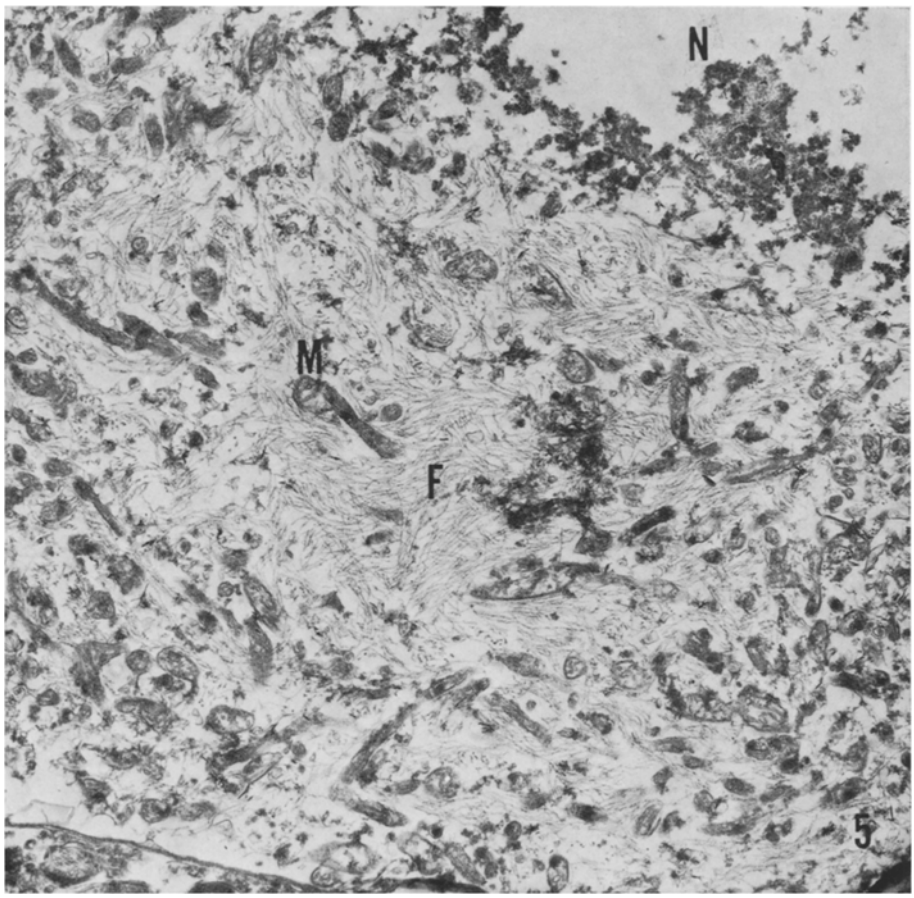


Fig. 5. Intracytoplasmic inclusion consisting of fine filaments (*F*) intermingled with degenerated mitochondria (*M*), comparable to argentophilic intracytoplasmic inclusion of Pick type seen under light microscope. *N* Nucleus. (×9600)

in diameter, whereas those in the peripheral zone, where they were loosely arranged, measured approximately 200 Å in diameter. These inclusions included no ordinary cytoplasmic organelles and were clearly demarcated from the rest of the cytoplasm, though they had no limiting membrane.

DISCUSSION

Our present case represents "unclassifiable presenile dementia" with clinical features of progressive dementia and muscular rigidity, the neuropathology of which revealed the presence of a great variety of "senile changes" in the C.N.S., including Lewy bodies and "Lewy-like-bodies". Nosologically, this case may represent either a combination of Alzheimer's disease, Pick's disease and idiopathic

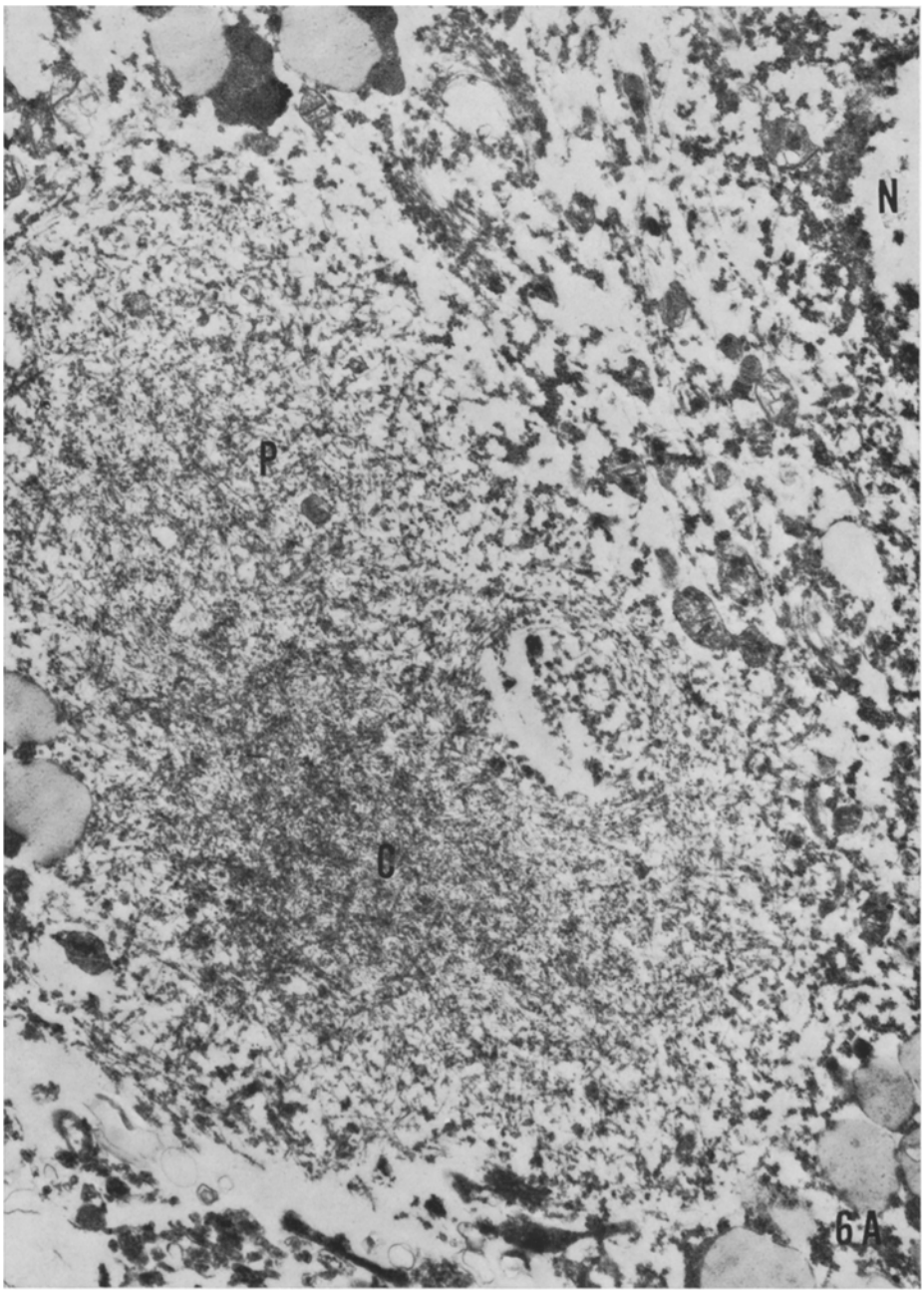
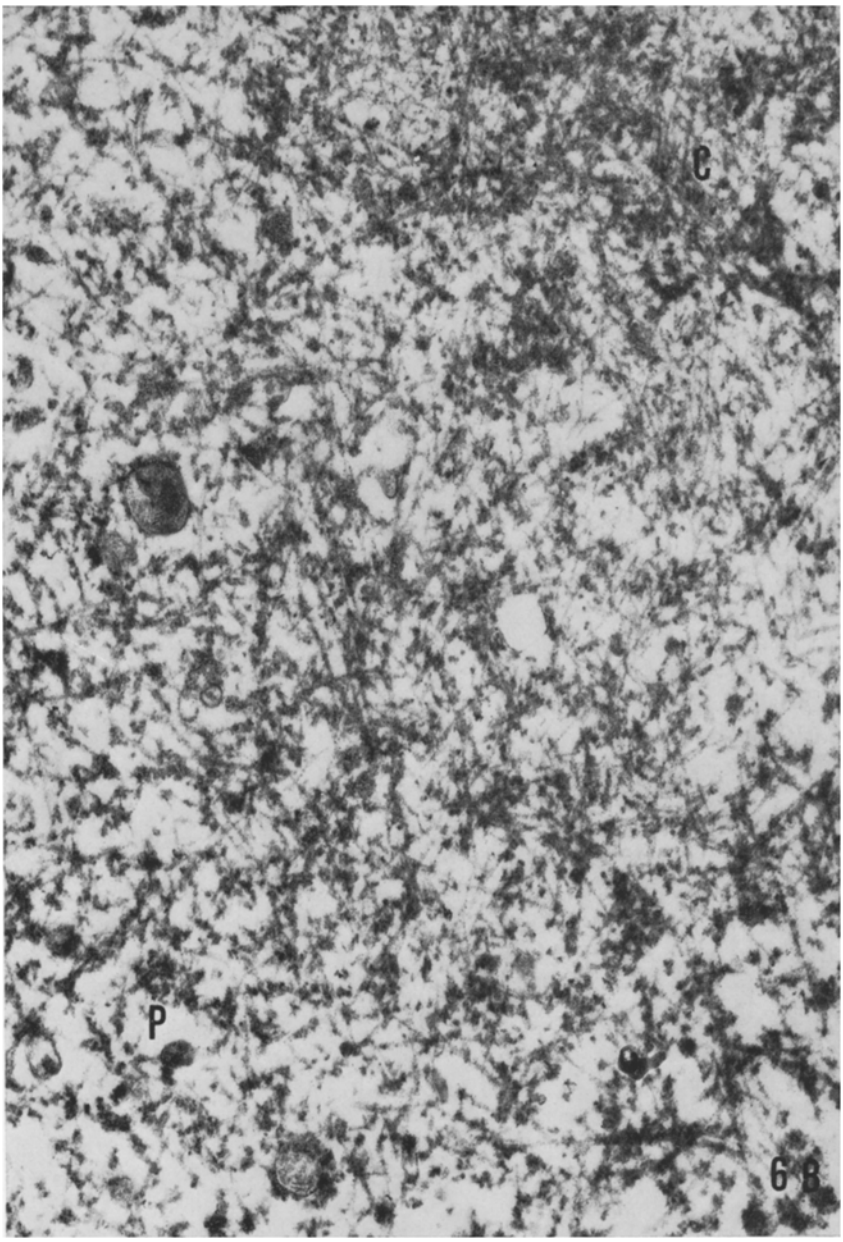


Fig. 6A and B. The ultrastructure of "Lewy-like-bodies" in the cerebral cortex. (A) The inclusion consists of fine meshwork of filamentous structures intermingled with fine dense granular materials. They are packed much denser in the central zone of the inclusion, forming a central denser core. *N* Nucleus, *C* central zone, *P* peripheral zone. (×10200). (B) Higher magnification of the inclusion. The filaments in the central zone measure approximately 120–130 Å in diameter, whereas those in the peripheral zones measure approximately 200 Å in diameter. Individual filaments are obviously different from neurofilaments in size and structure. *C* Central zone, *P* peripheral zone. (×24000)



Parkinsonism with "Lewy-like-bodies" in the cerebral cortices, or a single disease entity.

The most characteristic finding in this case was the presence of peculiar swollen cells with intracytoplasmic, eosinophilic and argentophilic inclusions ("Lewy-like-bodies"), which were widely scattered in the deep layers of all cerebral cortices. These intracytoplasmic inclusions were quite similar to Lewy bodies in light-

microscopic findings. Also the second type of neuronal inclusions revealed under the electron microscope was thought to represent Lewy bodies, because they were similar to one class of Lewy bodies described by Roy and Wolman (1969). Their ultrastructure was quite different from that of the first type of inclusions which consisted of neurofilaments and was thought to be comparable to Pick bodies.

Lewy bodies were first described by Lewy in the substantia innominata and the dorsal motor nucleus of the vagus in brains from patients with idiopathic Parkinsonism, though he errorneously equated these bodies to the "corpora amylacea" described in myoclonus epilepsy by Lafora (Lewy, 1912; Lafora, 1911). Trétiakoff first employed the eponym "Lewy body" in 1919. Subsequent studies by Hassler (1937, 1938), Greenfield and Bosanquet (1953), Lipkin (1959), den Hartog Jager and Bethlem (1960), Hamada and Ishii (1963), Forno (1969) and several others have established the distribution pattern and histochemical properties of Lewy bodies in Parkinsonism and related conditions. Ultrastructural studies have been reported by Duffy and Tennyson (1965) and Roy and Wolman (1969).

"Lewy-like-bodies" in the cerebral cortices of our case showed some differences from typical Lewy bodies in the brain stem. Light-microscopically, they were less eosinophilic, less well demarcated, and had poorer halos than the typical Lewy bodies. Ultrastructurally, the filaments in the outer zones of them did not radiate and circular profiles were not demonstrated as described by Duffy and Tennyson in the typical Lewy bodies.

Several cases with Lewy bodies in the cerebral cortices have been reported. Lipkin (1959) found Lewy bodies in the cerebral cortices of a case with idiopathic Parkinsonism. Forno (1969) studied brains with incidental Lewy bodies in the brain stem, and found them in the cerebral cortices in 6 out of 50 examnied cases. She indicated that Lewy bodies in the cerebral cortices were often less characteristic than those in other locations; they lacked central cores and sometimes halos. However, no detailed description about the properties and distribution of Lewy bodies in the cerebral cortices was given. Two cases reported by Okazaki et al. (1961) were the most remarkable ones. They, 67 and 70 year-old males, were characterized by progressive dementia and quadriparesis-in-flexion without Parkinsonian stigmata. Abundant "intraganglionic cytoplasmic inclusion bodies (Lewy type)" were found not only in the brain stem, but also in the cerebral cortices of these cases. These inclusion bodies in the cerebral cortices were found mainly in the cytoplasm of the medium-sized nerve cells, and distributed evenly throughout the cerebral cortices. Histologically, they were similar to those found in the brain stem, but always lacked central cores. Okazaki et al. interpreted this form of inclusion bodies in cerebral cortices as an early stage in the evolutionary process of Lewy bodies. Ikeda et al. (1975) also expressed similar views on the cytoplasmic bodies in the nerve cells in cerebral cortices found in their own case. Hamada and Ishii (1962) also described the evolutionary process of Lewy bodies. Since histologic, histochemical, and/or ultrastructural characteristics of the inclusion bodies in the cytoplasm of nerve cells in cerebral cortices of our case closely resembled those of Okazaki et al. and Ikeda et al. cases, they may represent an immature stage of Lewy bodies. The distribution pattern of typical Lewy bodies in the diencephalon and brain stem in our case as well as in Okazaki et al.

Table 1. Unclassifiable cases similar to the present case

Authors (year of public.)	Clinical data					Neuropathological data						
	Age	Sex	Duration of illness (year)	Dementia	Parkinson. stigmata	Lewy body in cereb. cort.	Lewy body in brain-stem	Senile plaq.	Neuro-fib. tangle	Pick body	Subcort. gliosis	Localized cereb. atrophy
Okazaki et al. (1961) 1	69	M.	1	+2	0	+3	+3	+1	0	/	0	0
2	70	M.	?	+2	0	+3	+3	+1	0	/	0	0
Ikeda et al. (1975)	38	M.	8	0	+3	+3	+3	0	0	0	0	0
Present case	65	F.	9	+3	+2	+3	+3	+3	+3	+2	+2	H. (lt>rt)
Berlin (1949) 1	56	M.	10	+3	+2	/	/	+3	+3	+3	+3	T. (lt<rt)
2	61	F.	4	+3	0	/	/	+3	+3	+3	+3	T.P. (lt = rt)
Neumann (1949)	67	M.	5	+3	0	/	/	+2	+2	+2	+3	F.T. (lt>rt)
Seitelberger et al. (1958)	64	F.	more than 3	+3	0	/	/	+3	+3	0	+3	T.F. (lt>rt)
Ando et al. (1965)	59	F.	more than 11	+3	+2	/	/	+1	+3	+1	+3	T.F. (lt<rt) [a]
Tariska (1970) 1	68	M.	6	+3	/	/	/	0	+3	0	+1	F.I.>T. (lt>rt)
2	62	F.	5	+3	/	/	/	0	+3	0	+2	T.I. (lt = rt) [b]
Kosaka et al.(1973)	54	F.	7–8	+3	+2	0	+1	+1	+3	+2	+2	T. (lt>rt) [c]
Oyanagi et al. (1975)	59	M.	7	+3	+1	0	0	+3	+3	+2	+3	T. (lt<rt)
Miyoshi et al. (1975)	57	M.	4	+3	+1	/	0	0	+3	0	+3	T.F.I. (lt = rt) [d]
Yoshimura et al. (1975)	68	F.	3.5	+3	0	0	+1	+3	0	0	+3	T. (lt>rt)

[a] Combined with pseudocalcareous deposition in the pallidum.
[b] Combined with Fahr's disease and arteriosclerosis.
[c] Combined with Fahr's disease.
[d] Combined with pseudocalcareous deposition in the pallidum and striatum.

0 = Absent; +1 = mild; +2 = moderate; +3 = severe; / = not described; H. = Hippocampal region; T = Temporal lobe; F = Frontal lobe; P = Parietal lobe; I = Insule.

and Ikeda et al. cases, was identical with that seen in idiopathic Parkinsonism. What was the cause of the occurrence of "immature Lewy bodies" in nerve cells of cerebral cortices in these cases, in addition to typical Lewy bodies in their preferential localities in the brains of Parkinsonism, has yet to be clarified.

The combined occurrence of abundant "senile changes" in C.N.S. such as senile plaques, Alzheimer's neurofibrillary tangles, granulovacuolar bodies Hirano bodies, torpedoes and even Pick bodies, in addition to the above mentioned "Lewy-like-bodies" and typical Lewy bodies, was another characteristic feature or our case. The former findings corresponded to combined Alzheimer's and Pick's disease, since localized atrophy including cortical deterioration with many Pick's argentophilic bodies and subcortical gliosis in our case was similar to that of Towfighi's "early Pick's disease" (1972). Ultrastructural findings of Pick's argentophilic bodies in our case were also similar to those reported by Rewcastle and Ball (1968), Towfighi (1972) and Oyanagi (1974).

McMenemy (1963) regarded those cases published by Berlin (1949), which had concurrent characteristics of both Pick's and Alzheimer's diseases, as examples of "Pick's and Alzheimer's double disease". Tariska (1970) called similar cases "superimposed Pick's and Alzheimer's disease". Those cases published by Neumann (1949), Seitelberger and Jellinger (1958) and Ando et al. (1965) also seem to be similar ones. Kosaka et al. (1973) reported a case of "unclassifiable presenile dementia" with the findings of Fahr's disease in addition to those of Pick's and Alzheimer's diesases. Recently, Oyanagi et al. (1975), Yoshimura et al. (1975) and Miyoshi and Kamiya (1975) also reported cases of dementia with morphological features of Alzheimer's and Pick's diseases.

The main features of the above mentioned cases were summarized and compared with our case in Table 1. Such cases as listed in Table 1 do not fit into the traditional classification, and reports of such cases are increasing in number recently. Our case will help in the possible reclassification of presenile dementia or Parkinsonism.

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