

Article abstract—In 58 patients with progressive supranuclear palsy (PSP), 19 (32.8%) had CT, MRI, or autopsy evidence of a multi-infarct (MI) state. The clinical findings in the infarct syndrome were similar to idiopathic PSP. Five MI-PSP patients had had a stroke, four had focal dystonia, two had hemiparesis, and one had an intention tremor of recent onset. In contrast, only 5.9% (12.9% of those with CT or MRI) of 426 Parkinson's disease patients had evidence of strokes. One case of PSP studied pathologically was attributed to cerebral amyloid angiopathy.

NEUROLOGY 1987;37:570-576

Progressive supranuclear palsy and a multi-infarct state

Richard M. Dubinsky, MD, and Joseph Jankovic, MD

The combination of dementia, parkinsonism, and supranuclear ophthalmoparesis defines progressive supranuclear palsy (PSP).¹⁻⁴ Multi-infarct (MI) dementia and MI parkinsonism have been well characterized.⁵⁻¹⁰ All the cardinal features of PSP may also be caused by strokes; therefore, we studied the frequency of cerebrovascular disease in PSP.

Patients and methods. We studied 58 consecutive patients with PSP evaluated in the Movement Disorder Clinic, Baylor College of Medicine. All had (1) onset after age 40; (2) parkinsonian findings of bradykinesia, postural instability, gait disorder, axial rigidity, and minimal or no rest tremor; (3) pseudobulbar signs including dysarthria, dysphagia, and a loss of emotional control; and (4) marked vertical supranuclear gaze palsy.¹¹ Vertical ophthalmoparesis may be seen in other parkinsonian disorders and may be lacking in pathologically proven cases of PSP.^{12,13} However, the clinical diagnosis of PSP is difficult if there are no oculomotor signs, and we required an ophthalmoplegia (an inability to move eyes more than 15° from the horizontal) for the diagnosis of PSP. The oculomotor findings were confirmed by a neuro-ophthalmologist and recorded on a videotape. CT or MRI (0.35- to 0.5-tesla field strength) was performed in all PSP patients.

Results. Nineteen (32.8%) PSP patients had either clinical or radiographic findings consistent with cerebrovascular disease (tables 1 and 2). In five patients, there was history of stroke before the onset of PSP symptoms. The two groups, 19 with MI-PSP and 39 with idiopathic PSP, were comparable with respect to mean age and duration of symptoms (table 2).

CT was performed in 43 patients and MRI in 15. Nineteen patients (10 with CT and 9 with MRI) had imaging evidence of an MI state: small infarcts in the periventricular region in 12, midbrain and brainstem

infarcts in 9, infarcts in the internal capsule and centrum semiovale in 5, and basal ganglia infarcts in 4 (table 1, figure 1).

The PSP patients were compared with a control group of 426 patients with Parkinson's disease (PD). The prevalence of stroke in the PD patients was only 5.9% (25 of 426) ($p < 0.00001$ by χ^2) (figure 2). Neuroimaging studies were performed in 192 PD patients: 184 had CT, 5 MRI, and 3 both. Only 23 (12.0%) patients had evidence of one or more strokes. MRI showed one or more infarcts in only two patients and was normal in six. Two additional patients had clinical evidence of stroke (hemiparesis, homonymous inferior quadrantanopia), but they did not have CT or MRI. When these patients were included, the prevalence of documented cerebrovascular disease in the PD patients was 12.9% (figure 2). The 2.5-fold higher frequency of cerebrovascular disease in PSP than in this selected PD group was significant ($p < 0.001$ by χ^2).

In the PD population, data on stroke risk factors were available for 110 patients. Seventy patients (63.6%) had at least one risk factor for stroke, such as hypertension (diastolic BP over 90 mm Hg) in 52 (47.3%), prior stroke in 7, and atrial fibrillation in 2. Other possible risk factors were myocardial infarction or coronary artery bypass in six and artificial aortic valve in one.

Sixty percent of all PSP patients had hypertension. Of the idiopathic PSP patients, 10 of 16 (62.5%) had risk factors for stroke; 7 (43.7%) had hypertension, 2 had coronary artery disease, 2 had diabetes mellitus, and 1 each had atrial fibrillation and type IV hyperlipidemia. In the MI-PSP group, 14 had hypertension (74%), 5 had a history of stroke, and 1 each had atrial fibrillation and congestive heart failure.

The following patient is described because of unique features. Unless otherwise stated, all laboratory studies are normal.

From the Baylor College of Medicine, Department of Neurology, Houston, TX.

Presented in part at the thirty-eighth annual meeting of the American Academy of Neurology, New Orleans, LA, April 1986.

Received May 27, 1986. Accepted for publication in final form August 6, 1986.

Address correspondence and reprint requests to Dr. Jankovic, Department of Neurology, Baylor College of Medicine, One Baylor Plaza, Houston, TX 77030.

Table 1. Clinical features of 19 patients with progressive supranuclear palsy and a multi-infarct state

Pt no./ age at onset/ sex/duration of symptoms (yrs)	Dementia score*/ Hoehn-Yahr stage†	Stroke risk factors	CT or MRI findings
1/64/M/6	4/3	HTN, +FHx, stroke (1)	CT: Multiple infarcts, B IC, B CS, BS
2/54/F/7	2/3	HTN, stroke (1)	MRI: Multiple periventricular lacunes and R medulla
3/62/F/12	0/4	HTN, +FHx	CT: Mild atrophy, R parietal periventricular, R post. parietal cortex, L brachium pontis
4/65/M/8	2/4	HTN, a.Fib	CT: Moderate atrophy, infarcts: R parietofrontal, periventricular, and midbrain
5/62/M/10	2/3	HTN, strokes (3)	CT: Infarcts: B BG
6/62/F/2	2/4	None	MRI: Infarcts, L cerebral peduncle and superior colliculus periventricular multiple infarcts, atrophy of inferior vermis
7/64/M/4	2/3	HTN, CHF, strokes (3)	CT: Multiple BG and periventricular infarcts
8/74/F/4	1/3	HTN	MRI: Moderate atrophy, infarcts: L thalamus and multiple B subcortical
9/61/M/7	3/5	None	CT: Infarcts: R parietal cortex and periventricular
10/61/M/7	3/3	HTN, DM, stroke (1)	CT: moderate atrophy, infarcts: B periventricular, L BG, L CS
11/60/M/3	2/4	HTN	CT: Moderate atrophy, infarct L putamen
12/63/F/9	1/3	HTN	CT: Infarcts: head of R CN, ant. R parietal cortex, small falx meningioma
13/72/M/2	2/3	None	CT: Moderate atrophy, infarct L head of caudate nucleus
14/80/M/4	1/3	HTN, DM	MRI: Mild atrophy, infarcts: B periventricular and R IC.
15/74/F/4	0/3	HTN	MRI: Mild atrophy, infarcts: B periventricular, L temporal cortex, and L brachium pontis
16/77/M/2	0/5	None	MRI: Infarcts: B periventricular, R occipital lobe, and central pons
17/60/M/3	2/2	+ ANA	MRI: Infarcts: R extreme capsule, anterior perforated substance, R periventricular area
18/71/M/4	2/3	HTN	MRI: Infarcts: B periventricular and slight periventricular edema
19/66/F/3	0/3	HTN	MRI: Infarcts: B periventricular

* Dementia score: 0—normal; 1—impaired cognition, eg, difficulty with arithmetic or abstraction, normal memory; 2—mild to moderate impairment of cognition and memory, but preserved orientation; 3—severe impairment of cognition, memory, and orientation; 4—global dementia (impairment in all functions).

† Hoehn-Yahr 0 to 5 stage.¹⁴

HTN Hypertension.	DM Diabetes mellitus.	IC Internal capsule.
+FHx Positive family history of stroke.	+ANA Positive antinuclear antibodies.	CN Candate nucleus.
a.Fib Atrial fibrillation.	R Right.	CS Centrum semiovale.
CHF Congestive heart failure.	L Left.	BS Brainstem.
	B Bilateral.	BG Basal ganglia.
		Infarcts = abnormal density consistent with infarcts.

Case report. This 70-year-old hypertensive man first noted forgetfulness, unsteadiness of gait, and blurred vision at age 64. He had had persistent horizontal diplopia for 2 years. His mother died of stroke.

He had a severe global dementia (could not recall any of three objects after 2 minutes) and a low-volume hypokinetic dysarthria. There was moderate hypomimia, body bradykinesia, and axial rigidity. There was marked limitation of vertical

gaze to less than 15° from the horizontal, with skewed deviation and internuclear ophthalmoplegia. The gaze palsy was corrected by the oculocephalic maneuver. His gait was broad-based and slow. Tendon reflexes were increased throughout, but there was no clonus.

CT showed an area of decreased density in the midbrain, internal capsule, basal ganglia, and centrum semiovale (figure 3, A and B). He improved transiently on treatment with the

Table 2. PSP vs PD: multi-infarct state

Category	N	M/F	Age at onset	Duration
			(yrs)	(yrs)
			Mean ± SEM	Mean ± SEM
PSP	58	1.64	66.0 ± 1.18	4.84 ± 0.36
Without MI*	39 (67.2%)	1.60	66.8 ± 1.46	4.28 ± 0.44
With MI†	19 (32.8%)	1.71	65.9 ± 1.56	5.31 ± 0.67
PD	426	1.83	59.7 ± 1.54	7.64 ± 0.25
Without MI	401 (94.1%)	1.92	59.6 ± 1.70	7.58 ± 0.26
With MI‡	25 (5.9%)	1.36	60.7 ± 2.08	9.14 ± 1.33
PD no CT/MRI	234	1.86	58.6 ± 0.63	8.10 ± 0.35
Without MI	232 (99.1%)	1.87	58.6 ± 0.63	8.10 ± 0.35
With MI	2 (0.9%)	1.00	59.0 ± 10.0	7.50 ± 6.50
PD with CT/MRI	192	1.74	57.8 ± 0.84	7.13 ± 0.33
Without MI	169 (88.1%)	1.89	57.2 ± 0.94	6.86 ± 0.33
With MI	23 (11.9%)	1.18	60.8 ± 2.18	9.25 ± 1.39

* Idiopathic PSP patients.
† PSP patients have 5.2-fold ($p < 0.00001$) increase in stroke compared with PD patients and 2.4-fold ($p < 0.001$) increase when compared with only PD patients who had brain imaging studies.
‡ Includes two patients with clinical evidence of stroke, but no neuroimaging studies.

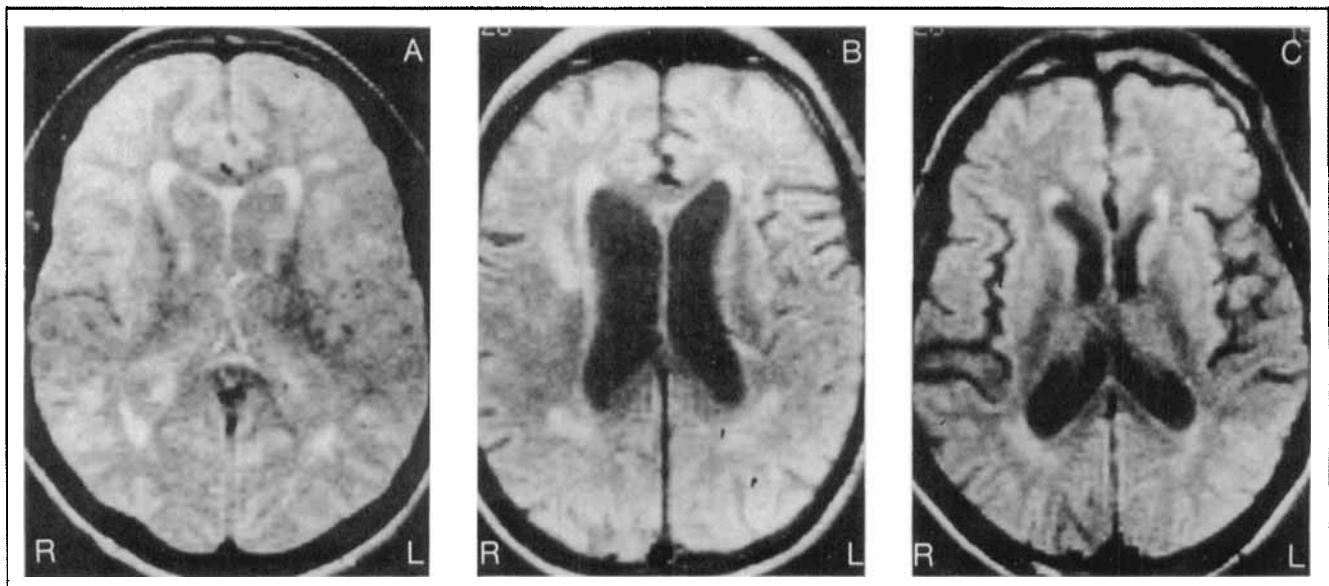


Figure 1. (A) MRI, patient no. 2, showing left frontal, right parieto-occipital, and frontal periventricular areas of high signal intensity consistent with infarcts. (Pulsing sequence, TR = 2.0 seconds; spin echo, TE = 56 msec). (B) MRI, patient no. 14, showing right internal capsule, right occipital, and bilateral periventricular with infarcts. (TR = 2.0 seconds, TE = 28 msec). (C) MRI, patient no. 16, showing periventricular areas of high signal intensity (TR = 2.0 seconds, TE = 28 msec).

combination of Sinemet and bromocriptine, but he died after an aspiration pneumonia.

At autopsy, the brain appeared to be slightly atrophic. Gross sections revealed several lacunae from 0.5 to 1.0 cm in diameter in the right centrum semiovale, substantia nigra, diencephalon, and pons. Microscopic examination showed infarcts associated with cerebral amyloid angiopathy (CAA) (figure 4, A and B). There were no neurofibrillary tangles, granulovacuolar degeneration, gliosis, neuronal cell loss, or other pathologic features typically found in idiopathic PSP.^{1,3}

Discussion. We found that PSP patients had a greater frequency of cerebrovascular disease (MI-PSP) than a

comparable population of PD patients ($p < 0.001$ by χ^2). Nineteen of 58 PSP patients (32.8%) had evidence of cortical, subcortical, or brainstem strokes, compared with 25 of 426 PD patients (5.9%). Besides the cardinal features of PSP, present in all patients, some also had apraxia of eyelid opening (levator inhibition), blepharospasm, horizontal gaze limitation, and internuclear ophthalmoplegia.^{11,15-17}

In the nine cases described by Steele et al,¹ there were no clues as to etiology, and vascular pathology was not noted. The typical findings included neuronal cell loss, gliosis, neurofibrillary tangles, and granulovacuolar degeneration in the pontomesencephalic tegmentum, tec-

tum, basal ganglia, vestibular nuclei, superior colliculi, periaqueductal gray matter, and the dentate nuclei.^{1,3} These pathologic findings were not seen in our autopsied patient, with an MI state, presumably due to CAA. That case initiated the comparative study.

Strokes have not been reported in studies of PSP. However, a case of clinically suspected MI state was reported to have pathologically documented features of PSP.¹⁸ Parkinsonism, sometimes spontaneously improving, has been attributed to multiple strokes in the basal ganglia.^{9,10} Two of our patients with MI-PD did not improve and remained the same. The progression seen in most of our MI-PSP patients could have been

due to the cumulative effects of recurring microinfarcts. Delayed-onset progressive motor disorders have been also attributed to axonal sprouting and other mechanisms.¹⁹

In addition to parkinsonism itself, other components of PSP (dementia,^{20,21} ophthalmoparesis,²²⁻²⁴ and dystonia²⁵) have each been seen in patients with multiple strokes. The unusual aspect of our cases was that all of these components were present. Because the patients had the clinical findings of PSP, they might have had idiopathic PSP with a coincidental MI state. Small infarcts do not always cause permanent clinical findings; specifically, infarction of the basal ganglia does not always result in parkinsonism.²⁶ While clinically similar, the 19 patients with MI-PSP may have had a syndrome distinct from the more common idiopathic PSP. In addition to the radiographic evidence of MI state and autopsy documentation in one case, some MI-PSP patients had a history of stroke (patients 2, 5, 7, 10, and 18), focal or segmental dystonia (patients 2, 5, 6, and 15), hemiparesis (patients 5 and 10), and a unilateral coarse intention tremor (patient 4).

Except for cerebral or brainstem atrophy, CT and MRI in idiopathic PSP are usually normal,^{11,27,28} and they are not usually indicated in typical PD. Thus, the 12.9% prevalence of stroke in the PD patients who had one of the neuroimaging studies is probably greater than the expected prevalence in the general PD population and may reflect a selection bias in favor of complicated PD. Therefore, the sixfold (2.4-fold when only patients with radiographic studies are compared) increase in the frequency of stroke in the PSP population over the PD group is probably an underestimation.

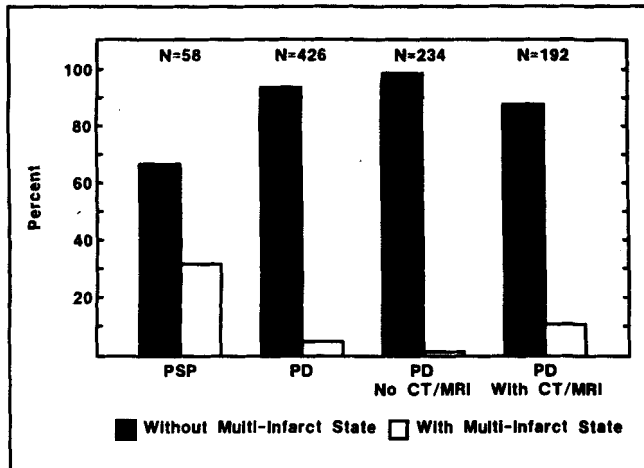


Figure 2. Comparison of PSP and PD patients with and without multi-infarct state.

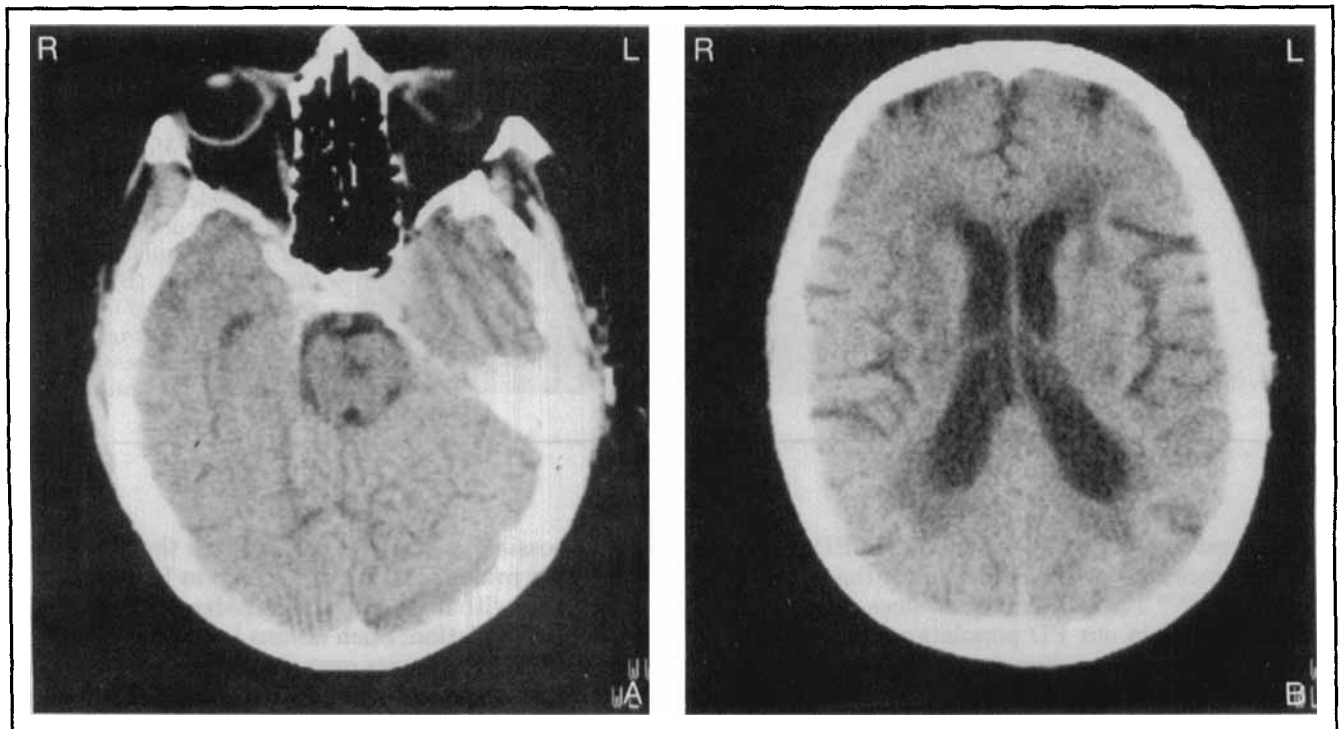
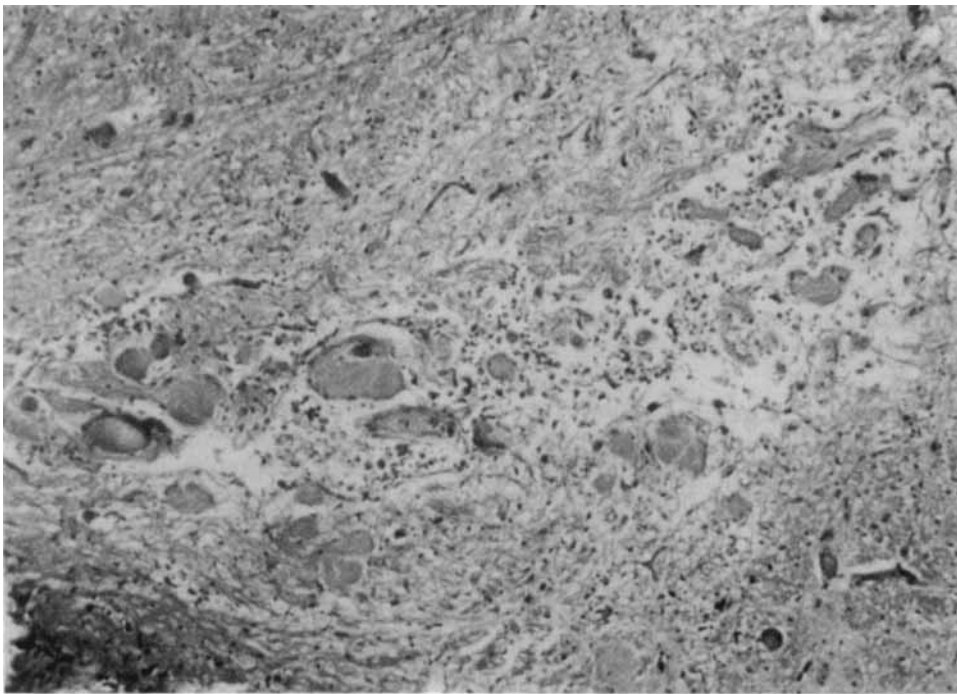
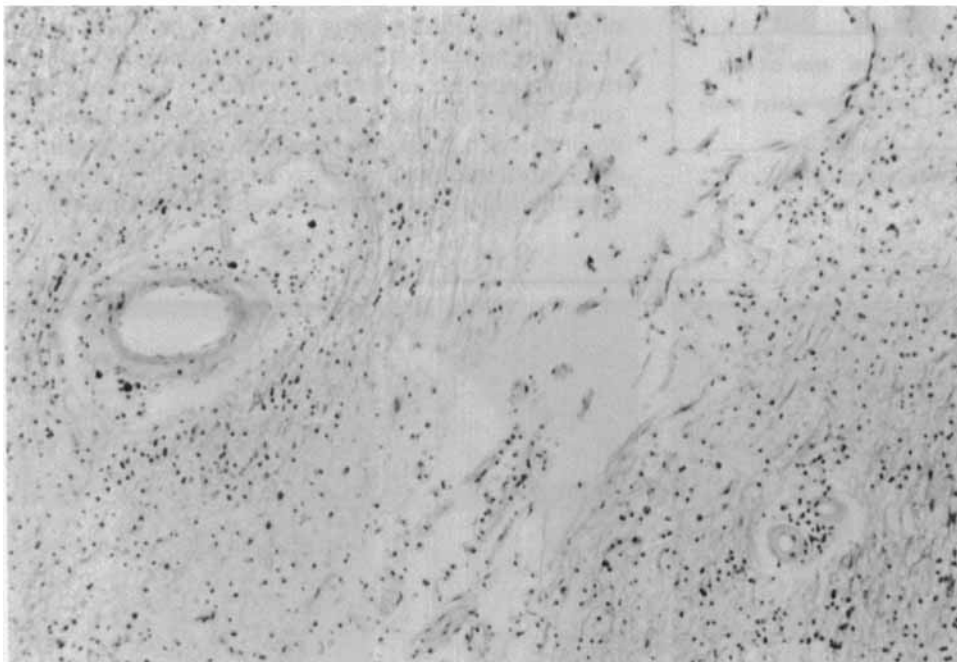


Figure 3. (A) CT through the midbrain of patient no. 1, showing an area of decreased density in the midbrain region. (B) CT through a higher level, showing bilateral periventricular lucencies, along with infarcts bilaterally in the internal capsule.



A



B

Figure 4. (A) Microscopic section (H-E stain) from the pontomesencephalic junction of patient no. 1 just inferior and lateral to the red nucleus. (B) Congo red stain of an old infarct in the pons showing thickened blood vessels with amyloid deposition.

In several community-based surveys of PD patients, the prevalence for stroke has been lower than expected, at 5.7 to 9%.^{29,30} These prevalence values are similar to the 5.9% found in our PD population. Therefore, the 32.8% prevalence of cerebrovascular disease in the PSP population is much greater than expected, and it probably reflects the high frequency of hypertension (60%), greater body weight, older age at onset, and other stroke risk factors.³¹

One possible criticism of our study is that the periventricular prolonged T2 lesions, seen in 12 (63.2%) of our patients with PSP, could be considered "normal" in an elderly population. Such lesions have been reported in up to 30% of patients over the age of 60.^{32,33} While pathologic correlation is lacking, these lesions probably are not "normal" and are associated with high incidence of hypertension, transient ischemic attacks, and cardiac disease.³² In one study, 78.5% of 151 adults older

than 50 years with periventricular high-signal intensity lesions had cerebrovascular risk factors, whereas only 7.8% without such risk factors had this MRI pattern.³⁴ While periventricular hyperintensity on spin-echo MRI is a common finding,³⁵ this is different from the patchy periventricular changes seen in the MI-PSP patients. Furthermore, only two of our patients (18 and 19) had such a periventricular change as the only MRI finding; all other patients had one or more focal areas of abnormal signal intensity. The abnormalities shown by both CT and MRI correspond to vascular lesions described in postmortem studies of MI states.³⁶

Dementia, gait disturbance, ataxia, and parkinsonism have been seen in patients with the subcortical arteriosclerotic encephalopathy (Binswanger's disease); however, the syndrome of PSP has not been described with this condition.³⁷⁻³⁹ CAA also has not been reported in patients with PSP. However, because CAA causes multiple small arteriolar infarcts and hemorrhages throughout the brainstem, basal ganglia, subcortical areas, and the cortex,⁴⁰ it is not surprising for this pathologic condition to cause the clinical syndrome of PSP.

We hypothesize that, in some patients with the clinical findings of PSP, subcortical hypoperfusion and frontal hypometabolism result in a loss of dopamine receptors in the striatum, nucleus accumbens, and the substantia nigra. Such receptor changes have been recently documented by *in vivo* positron tomography and by postmortem ligand binding studies in patients with PSP.⁴¹⁻⁴⁵

While clinically similar to PSP, the MI-PSP may be differentiated by prior strokes, the presence of focal dystonia, hemiparesis, and intention tremor. The neuroimaging studies (CT, MRI) show a much higher incidence of MI in PSP than in PD. However, autopsies will ultimately be needed to differentiate the MI-PSP from the idiopathic PSP.

Acknowledgments

We wish to thank C. Goodman, MD, Baylor College of Medicine, Department of Pathology, for the pathologic material; B. Jansson, PhD, University of Texas, Department of Biomathematics, M.D. Anderson Cancer Institute, for assistance with statistical analysis; N. Bryan, MD, PhD, Baylor College of Medicine, Department of Radiology, for reviewing the CTs and MRIs; and J. McCrary, MD, Baylor College of Medicine, Departments of Ophthalmology, Neurology, and Neurosurgery, for performing the neuro-ophthalmologic examinations.

References

1. Steele JC, Richardson JC, Olszewski J. Progressive supranuclear palsy: a heterogenous degeneration involving the brain stem, basal ganglia and cerebellum with vertical gaze and pseudobulbar palsy, nuchal dystonia and dementia. *Arch Neurol* 1964;10:333-359.
2. Jankovic J. Progressive supranuclear palsy, clinical and pharmacologic update. *Neurol Clin* 1984;2:473-486.
3. Kristensen MO. Progressive supranuclear palsy—20 years later. *Acta Neurol Scand* 1985;71:177-189.
4. Maher ER, Lees AJ. The clinical features and natural history of the Steele-Richardson-Olszewski syndrome (progressive supranuclear palsy). *Neurology* 1986;36:1005-1008.
5. Critchley M. Arteriosclerotic parkinsonism. *Brain* 1929;52:23-83.
6. Eadie MJ, Sutherland JM. Arteriosclerosis in parkinsonism. *J Neurol Neurosurg Psychiatry* 1964;27:237-240.
7. Schwab RS, England AC. Parkinson syndromes due to various specific causes, chap 9. In: Vinken PJ, Bruyn GW, eds. *Handbook of clinical neurology*, vol 6. Amsterdam: North Holland, 1968:227-247.
8. Parkes JD, Marsden CD, Rees JE, et al. Parkinson's disease, cerebral arteriosclerosis and senile dementia: clinical features and response to levodopa. *Q J Med* 1974;93:49-61.
9. Tolosa ES, Santamaria J. Parkinsonism and basal ganglia infarcts. *Neurology* 1984;34:1516-1518.
10. Friedman A, Kang UJ, Tatemichi TK, Burke RE. A case of parkinsonism following striatal lacunar infarction. *J Neurol Neurosurg Psychiatry* 1986;49:1087-1088.
11. Jackson JA, Jankovic J, Ford J. Progressive supranuclear palsy: clinical features and response to treatment in 16 patients. *Ann Neurol* 1983;13:273-278.
12. Guilloff RJ, George RJ, Marsden CD. Reversible supranuclear ophthalmoplegia associated with parkinsonism. *J Neurol Neurosurg Psychiatry* 1980;43:552-554.
13. Davis PH, Bergeron C, McLachlan DR. Atypical presentation of progressive supranuclear palsy. *Ann Neurol* 1985;17:337-343.
14. Hoehn MM, Yahr MD. Parkinsonism: onset, progression and mortality. *Neurology* 1967;17:427-442.
15. Troost BT, Daroff RB. The ocular motor defects found in progressive supranuclear palsy. *Ann Neurol* 1977;2:397-403.
16. Jankovic J. Apraxia of eyelid opening in progressive supranuclear palsy. *Ann Neurol* 1984;15:115-116.
17. Lepore FE, Duvoisin RC. "Apraxia" of eyelid opening: an involuntary levator inhibition. *Neurology* 1985;35:423-427.
18. Schainker BA. Autopsy diagnosis of progressive supranuclear palsy mistaken clinically for stroke: case report. *No Med* 1978;75:227-229.
19. Burke RE, Fahn S, Gold AP. Delayed-onset dystonia in patient with "static" encephalopathy. *J Neurol Neurosurg Psychiatry* 1980;43:789-797.
20. Cummings JL, Benson DF. *Dementia: a clinical approach*. Stoneham, MA: Butterworth Publishers, 1983:130-167.
21. Kaplan JG, Katzman R, Horoupian DS, Feld PA, Mayeux R, Hays AB. Progressive dementia, visual deficits, amyotrophy, and microinfarcts. *Neurology* 1985;35:789-796.
22. Jacobs L, Heffner RR, Newman RP. Selective paralysis of downward gaze caused by bilateral lesions of the mesencephalic periaqueductal gray matter. *Neurology* 1985;35:516-521.
23. Trojanowski JQ, LaFontaine MH. Neuroanatomical correlates of selective downgaze paralysis. *J Neurol Sci* 1981;52:91-101.
24. Buttner-Ennever JA, Buttner U, Cohen B, Baumgartner G. Vertical gaze paralysis and the rostral interstitial nucleus of the medial longitudinal fasciculus. *Brain* 1982;105:125-149.
25. Pettigrew LC, Jankovic J. Hemidystonia: a report of 22 patients and a review of the literature. *J Neurol Neurosurg Psychiatry* 1985;48:650-657.
26. Fisher CM. Lacunes: small, deep cerebral infarcts. *Neurology* 1965;15:774-784.
27. Ambrosetto P, Michelucci R, Forti A, Tassinari CA. CT findings in progressive supranuclear palsy. *J Comput Assist Tomogr* 1984;8:406-409.
28. Ambrosetto P, Bacci A. Unusual CT findings in progressive supranuclear palsy: case report. *J Clin Neuro Ophthalmol* 1985;5:59-61.
29. Marttila RT, Rinne VK. Arteriosclerosis, heredity, and some previous infections in the etiology of Parkinson's disease: a case control study. *Clin Neurol Neurosurg* 1976;79:46-56.
30. Kessler II. Epidemiologic studies of Parkinson's disease. III. A community-based survey. *Am J Epidemiol* 1972;96:242-254.
31. Van der Linden C, Jankovic J, Jansson B. Lateral hypothalamic dysfunction in Parkinson's disease. *Ann Neurol* 1985;18:137-138.
32. Bradley WG, Waluch V, Brant-Zawadzki M, Yadley RA, Wycoff RR. Patchy periventricular white matter lesions in the elderly: a common observation during NMR imaging. *Noninvasive Medical Imaging* 1984;1:35-41.
33. Bradley WG, Waluch V, Yadley RA, Wycoff RR. Comparison of CT and MR in 400 patients with suspected disease of the brain and spinal cord. *Neuroradiology* 1984;152:695-702.

34. Gerard G, Weisberg LA. MRI periventricular lesions in adults. *Neurology* 1986;36:998-1001.
35. Zimmerman RD, Fleming CA, Lee BCP, Saint-Louis LA, Deck MDF. Periventricular hyperintensity as seen by magnetic resonance: prevalence and significance. *AJNR* 1986;7:1-20.
36. Ishii N, Nishihara Y, Imamura T. Why do frontal lobe systems predominate in vascular dementia with lacunes? *Neurology* 1986;36:340-345.
37. Huang K, Wu L, Luo Y. Binswanger's disease: progressive subcortical encephalopathy or multi-infarct dementia? *Can J Neurol Sci* 1985;12:88-94.
38. Kinkel WR, Jacobs L, Polachini I, Bates V, Heffner RR. Subcortical arteriosclerotic encephalopathy (Binswanger's disease): computed tomographic nuclear magnetic resonance, and clinical correlations. *Arch Neurol* 1985;42:951-959.
39. Janota I. Dementia, deep white matter damage and hypertension; "Binswanger's disease." *Psychol Med* 1981;11:39-48.
40. Cosgrove GR, Leblanc R, Meagher-Villemure K, Ethier R. Cerebral amyloid angiopathy. *Neurology* 1985;35:625-631.
41. D'Antona R, Baron JC, Samson Y, et al. Subcortical dementia: frontal cortex hypometabolism detected by positron tomography in patients with progressive supranuclear palsy. *Brain* 1985;108:785-799.
42. Baron JC, Maziere B, Loc'h C, Sgouropoulos P, Bonnet AM, Agid Y. Progressive supranuclear palsy: loss of striatal dopamine receptors demonstrated in vivo by positron tomography. *Lancet* 1985;1:1163-1164.
43. Bokobza B, Ruberg M, Scatton B, Javoy-Agid F, Agid Y. [³H] Spiperone binding, dopamine and HVA concentrations in Parkinson's disease and supranuclear palsy. *Eur J Pharmacol* 1984;99:167-175.
44. Ruberg M, Javoy-Agid F, Hirsch E, et al. Dopaminergic and cholinergic lesions in progressive supranuclear palsy. *Ann Neurol* 1985;18:523-529.
45. Kish SJ, Chang LJ, Mirchandani L, Shannak K, Hornykiewicz O. Progressive supranuclear palsy: relationships between extrapyramidal disturbances, dementia and brain neurotransmitter markers. *Ann Neurol* 1985;18:530-536.

Neurology[®]

Progressive supranuclear palsy and a multi-infarct state

Richard M. Dubinsky and Joseph Jankovic

Neurology 1987;37:570

DOI 10.1212/WNL.37.4.570

This information is current as of April 1, 1987

Updated Information & Services	including high resolution figures, can be found at: http://www.neurology.org/content/37/4/570.full.html
Citations	This article has been cited by 3 HighWire-hosted articles: http://www.neurology.org/content/37/4/570.full.html##otherarticles
Permissions & Licensing	Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: http://www.neurology.org/misc/about.xhtml#permissions
Reprints	Information about ordering reprints can be found online: http://www.neurology.org/misc/addir.xhtml#reprintsus

Neurology® is the official journal of the American Academy of Neurology. Published continuously since 1951, it is now a weekly with 48 issues per year. Copyright © 1987 by the American Academy of Neurology. All rights reserved. Print ISSN: 0028-3878. Online ISSN: 1526-632X.

