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AJNR

CT scan in progressive supranuclear palsy.

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AJNR Am J Neuroradiol 1986, 7 (3) 529
<http://www.ajnr.org/content/7/3/529.citation>

This information is current as
of April 17, 2024.

Correspondence

CT Scan in Progressive Supranuclear Palsy

I read with great interest the paper by Masucci et al. [1] concerning the CT findings of 10 patients affected by progressive supranuclear palsy (PSP). The authors cited the case I had reported in 1981 [2]. In 1984 I reported CT findings of three patients with PSP, consisting of atrophy of the midbrain and quadrigeminal plate, with prominent interpeduncular, crural, ambient, and quadrigeminal plate cisterns, and dilatation of the aqueduct and posterior third ventricle [3]. At that time I emphasized the usefulness of the CT scan when the differential diagnosis between PSP and other extrapyramidal disorders is clinically difficult. It is gratifying to know that other authors obtained the same radiologic findings and drew the same conclusions without having read my paper. I believe the use of thin-section CT scans is not essential since I obtained the same results with a routine CT scan with a slice of 10-mm thickness. In 1985, I also reported finding in a patient affected by PSP [4] the same striking low-density midbrain abnormality extending from the interpeduncular cistern toward the aqueduct that was reported by Masucci et al. My patient was also affected by bilateral internuclear ophthalmoplegia. I suggested that this midbrain abnormality was probably due to degeneration of the midline midbrain structures, particularly of the medial longitudinal fasciculus. It would be of interest to know whether some of Masucci's patients who displayed this abnormality were clinically affected also by internuclear ophthalmoplegia. Further data correlated with pathologic specimens are needed to establish whether this midbrain abnormality is an artifact or represents a lesion.

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Reply

I wish to thank you for referring to me Dr. Paolo Ambrosetto's letter concerning our paper on PSP [1]. I would like to inform Dr. Ambrosetto that our study began in 1981 and the original paper was forwarded to the *AJNR* on December 3, 1983, and received on December 14, 1983 rather than 1984 as indicated in the journal. I have notified the editor of this error and the correction appeared in the November/December 1985 issue of *AJNR*, 6:980.

Since our paper was submitted for publication well before the 1984 publication date of Dr. Ambrosetto's paper and since I had discontinued our literature search well before December 1983, I feel this is sufficient explanation for my not being aware of his paper.

Concerning the low-density midbrain abnormality seen in six of our 10 patients, the statement in our paper that this abnormality had not been described before December 3, 1983, is still true.

This abnormality was first observed in one of our earliest scans in 1981. We considered the possibility of an artifact at that time. However, the appearance of a similar abnormality in five more cases suggested that the abnormality was not due to an artifact. I would have to disagree with Dr. Ambrosetto that this finding may be due to an artifact since our metrizamide cisternography study of three cases showed that the third ventricle was not involved and that the low-density abnormality was the result of the interpeduncular cistern invaginating the atrophic midline midbrain area. Internuclear ophthalmoplegia was not present in any of our patients.

I would have to agree that occasionally routine CT scans may reveal the atrophic changes described in PSP. However, our study was undertaken because routine posterior fossa scans in a number of our PSP patients had revealed little of significance.

I would like to add that not only did our study include 10 patients with PSP, it also included 31 control patients, 11 with Parkinson's disease, 10 with presumed Alzheimer's disease, and 10 with various cerebral, cerebellar, and brainstem disorders. Our intention was to determine how useful the CT findings in PSP were in the differential diagnosis of a number of neurologic disorders, particularly Parkinson's disease.

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