Invest Clin 35(2): 67 - 75, 1994

Antero-posterior localization of the pyramidal tract in the internal capsule in a living patient.

Fidias E. León-S.,[1] *Gustavo Pradilla A.*[2]

[1]Ryugakusei International Exchange Program, Clinical Neurology Unit, Third Department of Internal Medicine, 8-35-1. Sakuragaoka, Kagoshima 890, Japan, [2] Professor of Clinical Neurology, Ramon Gonzalez Valencia University Hospital, UIS-Santander University School of Medicine, Bucaramanga, Colombia.

Key words: Pyramidal tract, internal capsule, corticospinal pathway.

Abstract: A lacunar infarction occurred in the posterior limb of the internal capsule in a 33-years-old Colombian woman, showed an antero-posterior (A-P) course of the corticospinal pathway. Anatomical and histological studies had suggested this course, but to the author's knowledge, this could be the first reported case of a living patient with such features, confirming those previous reports about them.

Received: 31-01-94. Accepted:26-04-94.

INTRODUCTION

The pyramidal tract (PT) localization in the internal capsule (IC) has been discussed since the turn of the century. Pathological and microscopical studies have shown its A-P course, but a clinical correlation in a living patient had not been possible because of difficulties for searching such specific kind of lesions (16). Here, we present a case of cerebral embolism (CE) of paradoxical origen (PO) which allowed us to identify the A-P pathway of the PT. Thus, this case confirms macroscopic and microscopic descriptions about the pathway previously reported by others (9, 15).

CASE REPORT

A 33-years-old Colombian woman who was admited to the Ramon Gonzalez Valencia University Hospital in Bucaramanga, Colombia, presented sudden feeling of weakness and later felt senselessness. The length of that episode was unknown. She had aphasia for 12 hours

after the onset of her ictus. On admision her blood pressure was 90/60. Cardiac and lung examination were normal. She had varicous veins grade II/IV in the lower limbs (LL), mostly in the left side. The temperature of the left thigh was higher than that of the right side. It began to normalize after 24 hours. On neurological examination she was right-handed, and presented right central facial weakness, right hemiplejia and brisk tendon reflexes of right LL. At first evaluation both plantar responses were extensor but 4 days later it was only unilateral (right foot), and turned to be flexor 18 days after the ictus. Seven days after her stroke she could move her right foot, and 20 days later she could walk without assistance.

A lumbar puncture yielded cerebrospinal fluid (CSF) which was reported within normal limits (WNL). KOH, BK, Gram, VDRL, and India ink of CSF were negative. Prothrombin time and PTT were also normal.

Computarised Tomography (CT) of the head revealed a cerebral infarct in the posterior limb of the IC (Figs. 1 and 2). We didn't have access to Magnetic Resonance Imaging (MRI) facilities.

An echocardiography disclosed a patent foramen ovale (PFO) with a diameter of 6 mm. Nuclear angiography showed a valvular insufficiency of veins of right LL and a subsegmentary defect in perfussion at the posterobasal region of the lower lobe of the left lung.

She received anticoagulant therapy and was refered to the rehabilitation department with appropriate medical treatment.

DISCUSSION

This case fullfiled the clinical criteria suggested for Paradoxical Embolism (17), and presented the clinical characteristics of lacunar infarcts (11). The infarct was circumscribed to the posterior limb of IC of Central Nervous System (CNS). The clinical picture was in agreement with the localization of the infarct which involved the corticospinal pathway. A wider discussion on the neurological picture is presented elsewhere (10). We will comment here on the anatomical location and distribution of this vascular lesion of the PT.

It is known that the axons of the PT are formed by corticobulbar and corticospinal fibers and 60% of them arise from area 4. The remainder come from area 6 and the parietal lobe (3, 13). Its neurophysiologic relations within both brain and spinal cord have been described elsewhere (1).

The distribution of the PT in the IC has been discussed (2, 7) since the times of Charcot, Probst, Dejerine; and Marie & Guillain until a few years ago. Clinical and pathological correlations have shown that the PT can be located in the posterior portion of the IC (4, 5, 7). Ross (15) published a pathological study demonstrating the anteroposterior

Fig. 1a. CT scan of the head in the horizontal plane at high thalamic level showing the infarct at the anterior two thirds of the posterior limb of the IC (arrow).

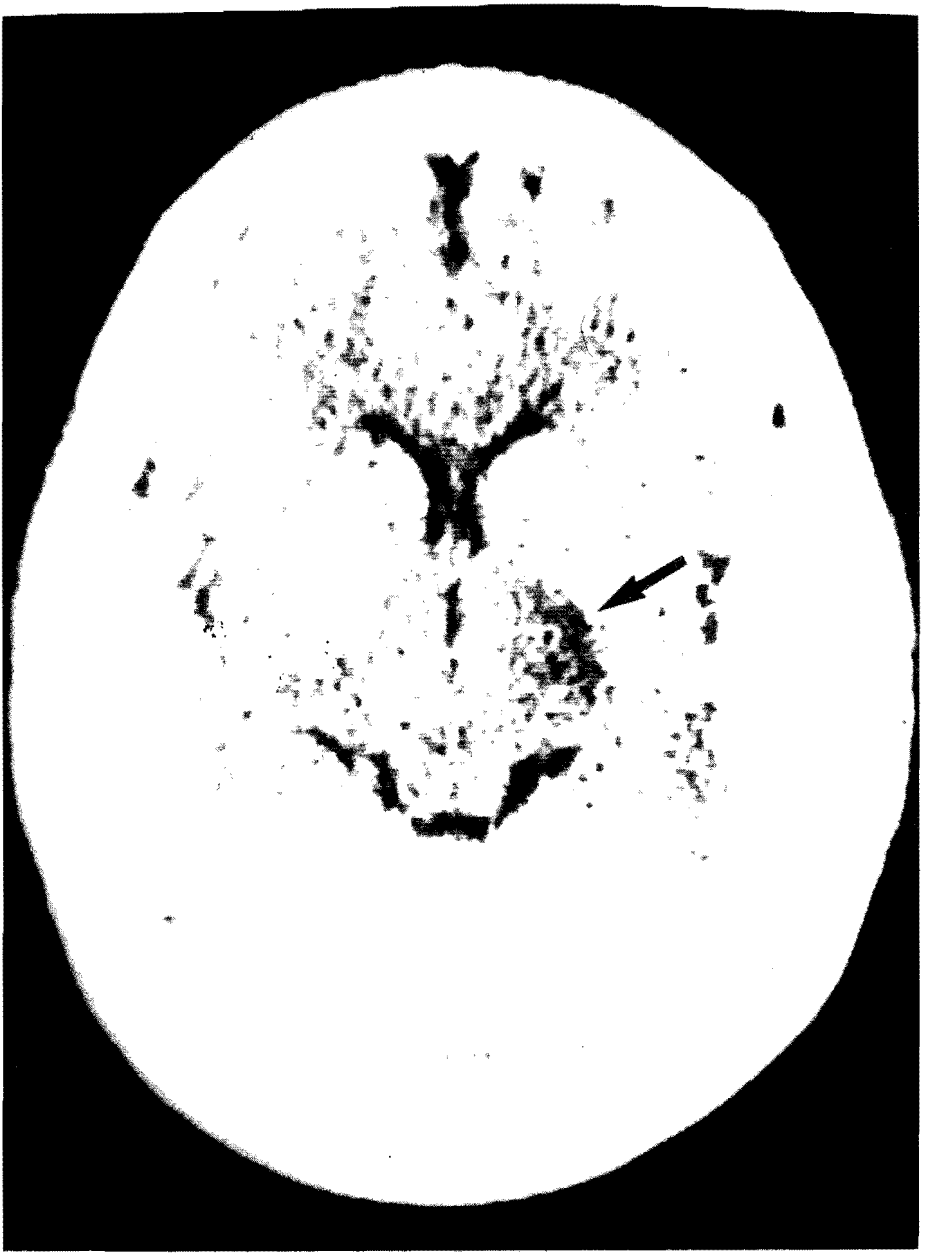

Fig. 2a. CT scan of the head in the horizontal plane at low thalamic level showing the infarct at the posterior third of the posterior limb of the IC (arrow).

course of PT through the IC. Clinical and microscopic correlations about it were suggested by Hanaway et al (8).

After Ross paper, an interesting computarised tomographic study performed on 18 patients was published (18). These patients presented lesions restricted to the IC with different localizations of their infarcts within it. From that study the authors concluded that the PT followed the traditional pathway but individual anatomical variations could be possible.

In 1988, Kretschmann published a very elegant microscopic study about the localization of the PT in the IC in man (9). His studies confirmed the AP course of the PT described previously by Ross (15).

Now, we present here, a clinical-neuroradiological case that coincides with the AP course of the PT descibed in those previous reports commented above.

The CT scan in figure 1a shows an infarct at high thalamic level. The PT entered the rostral IC at the anterior two thirds of the posterior limb. Then, the tract shifted its position to the dorsal third of the posterior limb of the IC, at low thalamic level as it is shown in the figure 2a.

These CT scans and its corresponding line drawing (Figure 1b and 2b), can be compared grossly with Ross and Kretschmann's studies (9, 15) as follows: our figure 1 can be compared with Figure 2b of Ross (15) and with Figures 2a and 2b of Kretschmann (9). Likewise, our Figure 2 can be correlated with the Figure 2d of Ross (15) and with the Figures 2c and 2d of Kretschmann (9).

Furthermore, we did several CT scans at different levels in the horizontal plane of the head of this patient, and we didn't find any other lesion either in the PT or in other part of her cerebrum (data not shown). Thus, based on the previous correlation we concluded that the PT followed an AP distibution through IC in this case.

MRI of the brain can be more informative than CT scan. Unfortunately in the place where this patient was diagnosed, this facility was unavailable. MRI also can detect wallerian degeneration secondary to infarctions of the PT which may occur within 3 months after the ictus (12). T-2 weighted coronal imaging has proved to be the best procedure for detecting the PT and its degeneration (12).

On the other hand, with Magnetic Stimulation (MS) the unaffected contralateral PT has been stimulated and it has been suggested that the lesions can be bypassed either by the corticoreticular tract which is bilateral in animals, or by axons that could have survived within the injured PT (6).

The recovery of patients with restricted lesions within the PT can be good (6), but the prognosis may be poor if any motor disability appears at clinical examination and also, if the capsular lesion is detected by means of MRI (14).

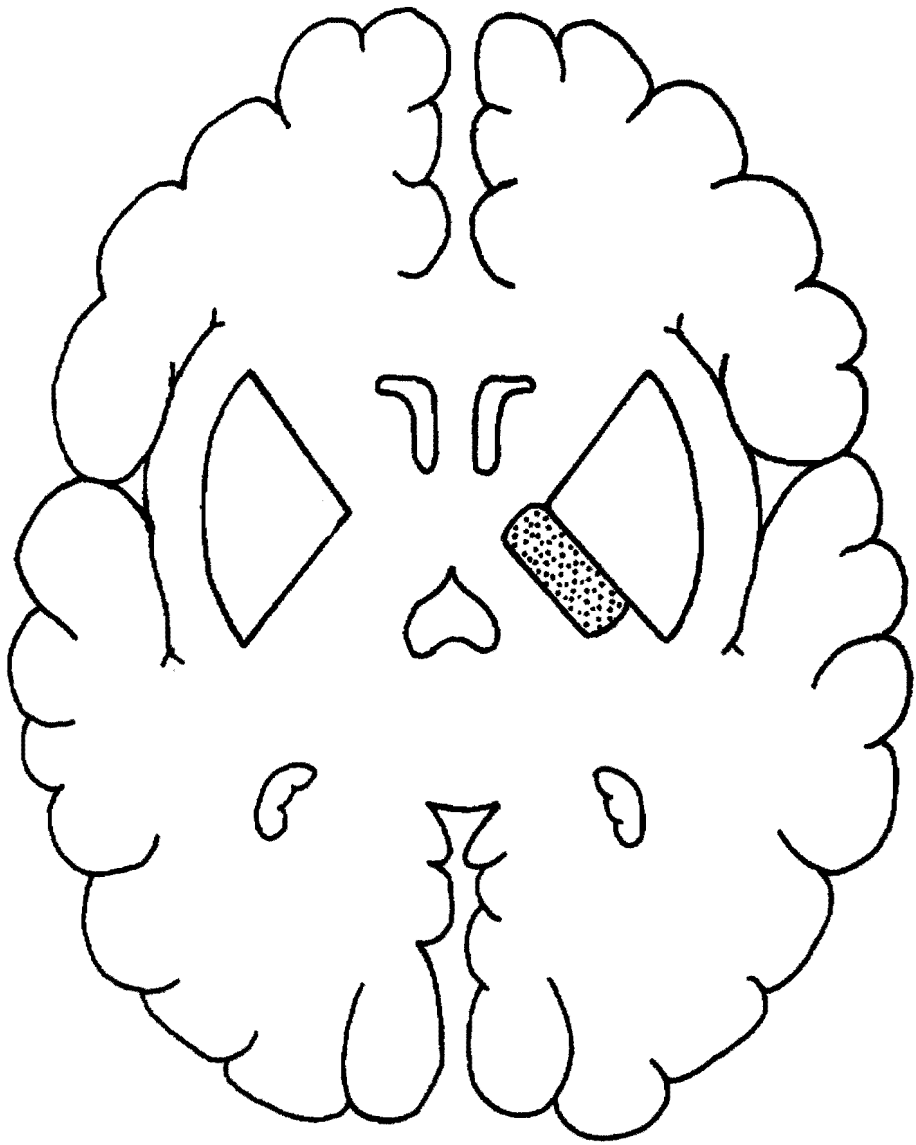

Fig. 1b. Line drawing showing more clearly the anterior localization of the infarct in the posterior limb of the IC.

Fig. 2b. Line drawing showing more clearly the posterior localization of the infarct in the posterior limb of the IC.

ACKNOWLEDGMENTS

The authors thank to Amparo Ariza-DeLeon, Ph.L., Saira S., M.B.B.S., and Neil Jhonson, B.S., for reviewing the English version of this manuscript and helpful comments; Dr. Alicia Rey for doing the neuroradiological study. One of us (F.E.L-S) thanks Dr. Mitsuhiro Osame for his guidance and support. A substancial part of this work will be submitted as dissertation for the Ph.D. degree in Clinical Neurology and Neurophysiology by Fidias E. Leon-S., to the Faculty of Medicine, University of Kagoshima.

RESUMEN

Localización antero-posterior del tracto piramidal en la cápsula interna de un paciente vivo. León-S., F.E. (Ryugakusei International Exchange Program Clinical Neurology Unit, Third Department of Internal Medicine 8-35-1 Sakuragaoka, Kagoshima 890, Japan), Pradilla-A., G. *Invest Clin 35(2): 67 - 75, 1994.*

Palabras claves: Tracto piramidal, capsula interna, vía cortico-espinal.

Una paciente colombiana de 33 años de edad, presentó un infarto localizado en el brazo posterior de la capsula interna. Este infarto nos permitió reconocer el curso antero-posterior del tracto piramidal. Estudios anatómicos e histológicos habían sugerido esto, pero, a conocimiento de los autores, este podría ser el primer caso clínico-radiológico descrito con tales hallazgos, confirmando así los informes hechos previamente.

REFERENCES

1- CAMBIER J.: Le faiceau pyramidal: pour qoui faire?. Neurologia 1:229-231, 1986.

2- COLE M.: Internal capsule. Neurology 40:1480, 1990.

3- DAVIDOFF R.A.: The pyramidal tract. Neurology 40:332-339, 1990.

4- ENGLANDER R.N., NETSKY M.G., ADELMAN L.S.: Localization of human pyramidal tract in the interna capsule: anatomic evidence. Neurology 25:823-826, 1975.

5- FERRERO J., TIRADOS F.: Hemiacroplejia proporcional y localización del haz piramidal en la capsula interna. Neurologia 6:71-73, 1991.

6- FRIES W., DANEK A., WITT T.N.: Motor responses after transcranial electrical stimulation of cerebral hemispheres with a degenerated pyramidal tract. Ann Neurol 29:646-650, 1991.

7- HANAWAY J., YOUNG R.R.: Localization of the pyramidal tract in the internal capsule of man. J Neurol Sci 34:63-70, 1977.

8- HANAWAY J., YOUNG R.R., NETSKY M., ADELMAN L.: Localization of the pyramidal tract in the internal capsule. Neurology 31:365-366, 1981.

9- KRETSCHMANN H.J.: Localization of the corticospinal fibers in the inter-

nal capsule in man. J Anat 160:219-225, 1988.

10- LEON-S F.E., PRADILLA G.: Embolismo cerebral de origen paradójico en Colombia: Primer caso diagnosticado en un paciente vivo. Acta Neurol Col 9:231-239, 1993.

11- ORGOGOZO J.M., BOGOUSSLAVSKY J.: Lacunar syndromes en: Hand-book of Clinical Neurology, vol. 54. Vascular Diseases Part II. pp. 235-269. Toole J.F., ed. Elsevier, Amsterdam, 1989.

12- ORITA T., TSURUTANI T., IZUMIHARA A., MATSUNAGA T.: Coronal MRI imaging for visualization of wallerian degeneration of the pyramidal tract. J Comp Ass Tomogr 15:802-804, 1991.

13- PERCIAVALLE V.: Relationships between pyramidal and extrapyramidal system. Boll Soc Ital Biol Sperim 11:1395-1400, 1986.

14- PUJOL J., MARTI-VILLATA J.L., JUNQUE C., VENDRELL P., FERNANDEZ J., CAPEDVILLA A.: Wallerian degeneration of the pyramidal tract in capsular infarction studied by magnetic resonance imaging. Stroke 21:404-409, 1990.

15- ROSS E.D.: Localization of the pyramidal tract in the internal capsule by whole brain disection. Neurology 31:59-64, 1980.

16- ROSS E.D.: Localization of the pyramidal tract in the internal capsule (Reply). Neurology 31:366-367, 1981.

17- RYDEN-JONES Jr., H., CAPLAN L.R., COME P.C., SWINTON N.W., BRESLIN D.J.: Cerebral embolism of paradoxical origin. Ann of Neurol 13:314-319, 1983.

18- TREDICI G., PIZZINI G., BOUGLIUN G., TAGLIABUE M.: The site of motor corticospinal fibers in the internal capsule of man. A computarised tomographic study of restricted lesion. J Anat 134:199-208, 1982.