Severe Unilateral Proprioceptive Loss in Medullary-rostral Spinal Cord Infarction. A Posterior Spinal Artery Syndrome

Case Report

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Abstract— Severe Unilateral Proprioceptive Loss in Medullary-rostral Spinal Cord Infarction. A Posterior Spinal Artery Syndrome

Introduction. The symptoms and signs found in patients with lateral medullary infarction have been described for more than 200 years. The major symptoms are vestibulo-cerebellar and dysphagia and dysphonia attributable to structures innervated by the ambiguous nucleus. Sensory symptoms and signs are often present in the ipsilateral face and contralateral limbs and trunk and sometimes the face. **Case Report.** A 29 year old woman had headache and left neck pain during weeks. Her left side suddenly became "completely dead and lacked feeling." She saw double and was dizzy. Her right side remained normal. MRI showed infarction in the left lateral caudal medulla extending into the medulla-spinal junction. Vascular imaging showed a dissection of the left vertebral artery . Second case is A retired man with his neurological symptoms and signs developed in steps. He first noticed while shaving that his left face was numb, A week later, while showering, he became unsteady and had discomfort behind his left ear and in his left neck. MRI showed an infarct limited to the caudo-medial portion of the left dorsolateral medulla that extended into the rostral spinal cord. **Discussion.** Loss of position sense in the limbs ipsilateral to a medullary infarct has only rarely been reported. Lesions of the vertebral artery just above or below dural penetration are most likely to cause medullary infarcts that are tegmental rather than basal and to cause rostral spinal cord infarction. This region of the vertebral artery is rarely affected by atherosclerosis and the commonest lesion is dissection. **Ictus 2020;1(1):e01092001008**

Keywords— Proprioceptive loss, Spinal cord infarction, Posterior spinal artery syndrome

Resumen— Pérdida Propioceptiva Severa en infarto medular rostral. Un Síndrome de la Arteria Espinal Posterior.

Introducción. Los síntomas y sigmos encontrados en pacientes con infarto bulbar lateral han sido descrito desde más de 200 años. La mayoría de los síntomas son vestíbulo-cerebelosos, disfagia y disfonía atribuidos a estructuras invervadas por el núcleo ambiguo. Los signos y síntomas sensoriales están presentes frecuentemente en la cara ipsilateral y extremidades y tronco contralateral y algunas veces la cara. **Caso Clínico.** Presentamos el caso de una mujer de 29 años con cefalea y dolor de cuello iquierdo de semanas de evolución. Súbitamente perdió la sensibilidad, presentó diplopia y mareo. Su ladoderecho era normal. La resonancia magnética mostró infartobulbar lateral izquierdo en la porción caudal, con extensión a la unión bulbo-espinal. La imagen vasculr mostró disección de la arteria vertebral izquierda. El segundo caso es un hombre retirado con síntomas proresivos. Primero notó adormecimiento de hemicara izquierda, una semana después notó inestabilidad y malestar atrás de la orea izquierda y cuello. La resonancia magnética mostró infarto en la porción caudo-medial de la región dorsolateral del bulbo raquídeo extendido a la región dorsal de la médula espinal. **Discusión.** La pérdida del sentido de posición en las extremidades ipsilateral al infarto bulbar solo se ha reportado rara vez. Esta región de la arteria verebral es poco afecada por ateroesclerosis y la causa más frecuente es disección arterial. **Ictus 2020;1(1):e01092001008**

Palabras clave— Pérdida propioceptiva, infarto de la médula espinal, síndrome de la arteria espinal posterior

INTRODUCTION

The symptoms and signs found in patients with lateral medullary infarction have been described for more than 200 years.[1–5] The major symptoms are vestibulocerebellar and dysphagia and dysphonia attributable to structures innervated by the ambiguous nucleus. Sensory symptoms and signs are often present in the ipsilateral face and contralateral limbs and trunk and sometimes the face. These regions have abnormal pain and temperature sensations related to dysfunction of the spinothalamic, quintothalamic, and spinal nucleus and tract of V. Ipsilateral sensory and proprioceptive limb abnormalities are rarely mentioned. Two reports describe sensory symptoms affecting the limbs ipsilateral to lateral medullary infarction but none of the patients reported were noted to have severe proprioceptive loss involving these limbs.[6,7] I (LRC) now describe two patients with infarction in the medulla extending into the rostral spinal cord who had severe proprioceptive loss in the limbs ipsilateral to the infarction.

CASE REPORT

Case 1 A 29 year old woman had headache and left neck pain during weeks. Her left side suddenly became "completely dead and lacked feeling." She saw double and was dizzy. Her right side remained normal. Eight weeks after her stroke, examination by LRC showed: torsional nystagmus with the left eye beating down on left lateral gaze. On right gaze she had small amplitude horizontal nystagmus. Her face was symmetric. Her left palate did not move well. Cold and pain sensation were diminished on the left face. Her limbs showed normal power. She had an intension tremor on left finger-to-nose, and toe to object testing. She had diminished appreciation of touch in the left arm and hand. She had very severe position sense loss involving the left limbs. She could not identify movements of the shoulder, arm, wrist, or hand. She had severe sensory ataxia when using the arm without visual control. Vibration sense was present but diminished in the left fingers and wrist. She could identify vibration in her left foot and movements of the left leg; position sense was lost in the left foot and toes. Cold and pain sensation was diminished in the right arm, trunk, and lower extremity.

MRI showed infarction in the left lateral caudal medulla extending into the medulla-spinal junction. Vascular imaging showed a dissection of the left vertebral artery within the V2 and V3 portions with decreased flow in the left intracranial vertebral artery. (Figure 1). The signs were attributable to: the spinal nucleus and or tract of V, the spinothalamic tract, the inferior cerebellar peduncle and left cerebellum, and nuclei and or tracts at the medulla-spinal junction that convey proprioceptive information from the ipsilateral limbs.

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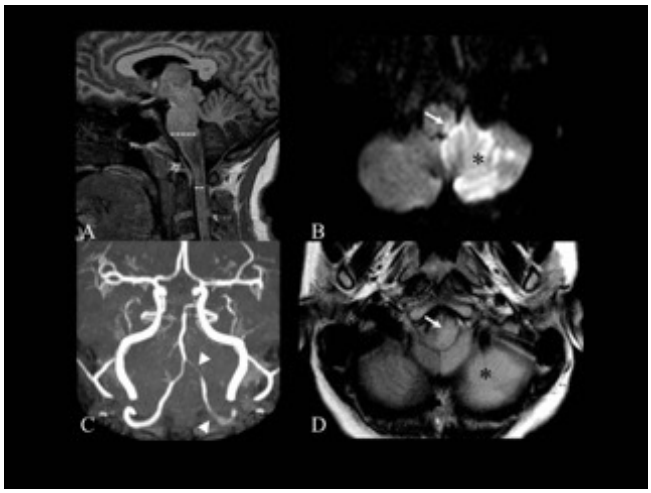

Figure 1: Left para-sagittal noncontrast T1 image (A) showing hypointense signal in the medulla and rostral cervical spinal cord at the C1 level (white dashed lines) indicating the full extent of the infarct. Representative DWI (B) and FLAIR (D) images through the lower medulla (white arrow) and lower cerebellar hemisphere (*) show acute left-sided infarcts. (C) Noncontrast 3D TOF MRA with loss of flow void and narrow caliber of the left V3 and V4 segments compatible with dissection (white arrowheads).

When examined 2 years after the stroke, proximal position sense in the left arm had improved. If the left arm was moved into various positions, with her eyes closed, she could point with her right index finger to the position of her left thumb. She still could not identify movements of the left wrist or fingers. Position sense was still lost in the left toes. She perceived vibration in the left fingers and toes. Gait was improved.

Case 2 A retired man with a long history of hypertension was seen by LRC 9 weeks after his stroke. His neurological symptoms and signs developed in steps. He first noticed while shaving that his left face was numb. Pain above his left eye lasted about 10 minutes. He noticed no limb difficulty. A week later, while showering, he became unsteady and had discomfort behind his left ear and in his left neck. His left face was numb and his gait was uncoordinated, but he had no important limb dysfunction. He noticed that he could not tell the location of his left arm. He found his arm in various positions and his arm and hand were very uncoordinated when moved. He could not walk. He became aware that he had difficulty feeling cold or hot temperatures on his right leg.

On examination: his pupils were 3 mm and reacted normally; eye movements were normal without nystagmus. His facial movements were symmetric; cold was felt less well on the left face. He held his left arm flexed and with his eyes closed, he had no idea where the arm or hand were located. Position sense was absent in the left hand and he did not feel vibration at the fingers but did feel cold. Strength was preserved, although he had difficulty directing his left arm in space. Finger to nose testing with his left arm was clumsy with eyes open but with eyes closed it was hopeless. He could not get anywhere near his nose and could not feel where his arm was in space. No deficit was detected in the right arm with touch, position or vibration. He had decreased pain and temperature

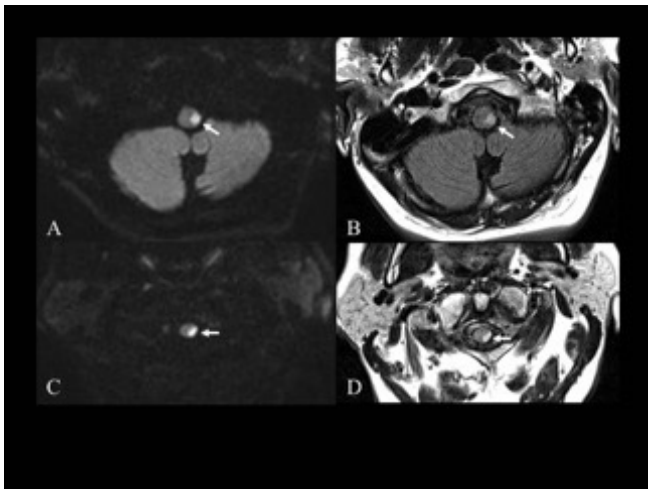

Figure 2: Axial DWI image through the lower medulla (A) and upper cervical cord at the C1 level (C) with corresponding FLAIR images (B and D respectively) showing left-sided acute infarcts, as indicated by the white arrow.

sensation in his right limbs and trunk. He had a very severe loss of position and vibration sense in the left leg. Position sense and vibration were preserved in the right foot. Reflexes were brisk bilaterally and plantar responses were extensor. When he stood, he kept his knees bent and tried to walk, but he could not tell where his left leg was located.

MRI showed an infarct limited to the caudo -medial portion of the left dorsolateral medulla that extended into the rostral spinal cord. (Figure 2). Vascular imaging did not show stenosis or other definite vertebral artery abnormality. The abnormalities found on exam were attributable to involvement of structures in the left lateral medulla: the spinal nucleus and or tract of V, the spinothalamic tract, the inferior cerebellar peduncle, and nuclei and/or tracts at the medulla-spinal junction that convey proprioceptive information from the ipsilateral limbs.

When examined 6 months after the stroke, position sense loss was still severe in the left arm, wrist and fingers. He could now detect motion of his left toes and foot and could bear weight better. He used a walker or cane and walked with great effort.

Discussion

Loss of position sense in the limbs ipsilateral to a medullary infarct has only rarely been reported. Kim et al analyzed ipsilateral limb sensory symptoms and signs in 12 patients (6.7%) among 174 individuals who had lateral medullary infarcts.[6] These patients all described paresthesias usually characterized as numbness. Three patients noted a tight or aching sensation over the upper arm and trunk. The sensory symptoms were limited to the hand in five patients, the upper extremity in one, the arm and upper trunk in one, and the whole hemibody/limbs in five. Sensory symptoms were most severe in the fingertips except in one patient, who had an uncomfortable aching sensation in the proximal arm. Vibration sense was decreased in the involved limb in six patients, and proprioceptive sensation was impaired in four. The objective sensory deficit was usually slight. The infarcts in these patients involved the caudal dorsomedial medulla as in the 2 patients in this report.[6]

Chen and colleagues recently reported two patients with cervical vertebral artery dissections who had spinal cord infarcts in the territory of the posterior spinal artery.[7] One of the patients had bilateral dorsal cord infarcts, the other a unilateral infarct. In one patient “deep feeling and tactile sensations on both sides were weak.” In the other patient “disturbances of touch and temperature sensation on the lower limb” were noted. There were no comments on examination of position and vibration sense. The authors reviewed 22 reports that discussed 28 patients with infarcts in the rostral spinal cord. Almost all had cervical vertebral artery dissections and the discussions centered on the radiological features of the dissections and the brain imaging. Sensory and proprioceptive examination findings in this report and in cases reviewed from the literature are not described. None were noted to have severe position sense loss in an arm.

The cuneate and gracile fasciculi that carry proprioceptive information from the limbs travel in the posterior columns of the spinal cord. (Figure 3) The gracile fasciculus contains nerve fibers from the lower extremities and the lower trunk; the sensory fibers from the arm and upper trunk in the fasciculus cuneatus are organized so that the hand area is located most laterally and the trunk area more medially. The fibers in these tracts synapse in the cuneate and gracile nuclei located dorso-medially in the caudal medulla and in the rostral spinal cord. Fibers emanating from these nuclei ascend as internal arcuate fibers to enter the medial lemniscus which travels ipsilaterally until its decussation more rostrally in the medulla than the pyramidal decussation. The infarcts in these 2 patients must have involved neurons in the gracile and cuneate nuclei or the arcuate fibers emanating from these nuclei that course to the medial lemniscus. If scattered neurons in the nuclei were infarcted and many spared, recovery of proprioceptive functions would be posited to improve more than if internal arcuate fibers were permanently damaged. Fibers are in general more resistant to infarction than nerve cells. Severe unilateral proprioceptive loss is a marker for rostral spinal cord involvement.

The blood supply of the most rostral portion of the lateral aspect of the spinal cord arises from the posterior spinal artery.[8,9] This vessel may originate from either the extradural or intradural portion of the vertebral artery as it enters the cranium or from its posterior inferior cerebellar artery branch. A branch of the posterior spinal artery supplies the caudal portion of the restiform body and the gracile and cuneate tubercle regions. The branching of this artery and the supply areas are variable and not studied or described in detail. Lesions of the vertebral artery just above or below dural penetration are most likely to cause medullary infarcts that are tegmental rather than basal and to cause rostral spinal cord infarction. This region of the vertebral artery is rarely affected by atherosclerosis and the commonest lesion is dissection.

In the first patient the vertebral dissection caused an occlusion of the vertebral artery in the region that gives rise to

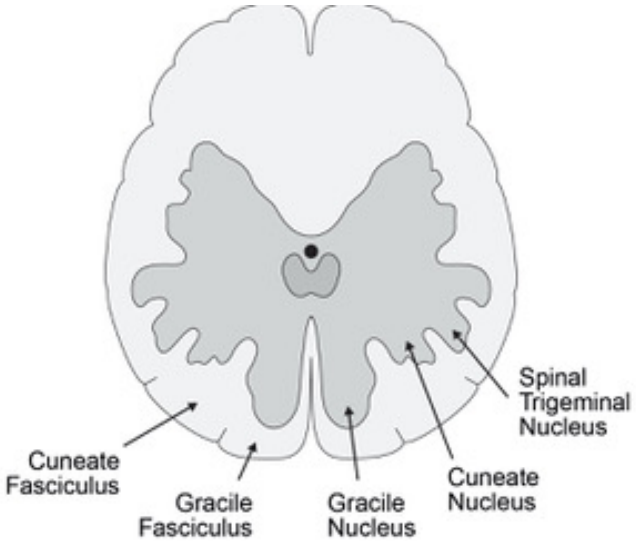

Figure 3: . Drawing that shows nuclei and tract in the rostral spinal cord.

the posterior spinal artery. The second patient had a long history of hypertension, and had a course of development of the infarct during a week. He most likely had an atheromatous branch occlusion affecting arteries that penetrate the lateral medullary fossa including the posterior spinal artery or a dissection that was not evident in vascular imaging. Most previously reported patients with posterior spinal artery territory infarcts have had vertebral artery dissections.

Patients with pontine, midbrain, and even thalamic infarcts do not have severe unilateral or bilateral proprioceptive loss. Patients with lesions limited to the medulla oblongata do not have severe unilateral proprioceptive loss. The presence of an upper limb severe sensory proprioceptive loss probably localizes the lesion to the high rostral spinal cord in the region of the cuneate nucleus or fascicle. When the clinical findings and /or imaging are those of lateral medullary infarction, severe unilateral proprioceptive loss indicates extension of the infarct to the rostral posterior-medial spinal cord and makes vertebral artery dissection, the most likely etiology.

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