Radiology Case Reports 17 (2022) 4148–4151

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/radcr

Case Report

Ocular contrapulsion followed by ipsipulsion in Wallenberg syndrome: The first case report in literature ☆

Euldes Mendes Junior[a], *Fernando Magri*[b], *Ana Beatriz Ramalho Leite Silva*[c], *Guilherme Pinheiro Diógenes*[c], *Marina Feitosa de Castro Aguiar*[c], *Lucas Bessa*[i], *Luciano Barroso de Albuquerque Filho*[c], *Sara Diógenes Peixoto de Medeiros*[h], *Luiz Aldir da Silva*[f], *Tiago Antoniol*[g], *Leandro Freitas Oliveira*[d,e,f], *Júlio César Claudino dos Santos*[d,e,f,*]

[a] *Universidade Federal de Minas Gerais, Belo Horizonte, MG, Brazil*
[b] *Universidade de São Paulo, São Paulo, SP, Brazil*
[c] *Faculdade de Medicina, Centro Universitário Christus, Fortaleza, CE, Brazil*
[d] *Universidade Federal de São Paulo, São Paulo, SP, Brazil*
[e] *Laboratório de Neurociências, Departamento de Neurologia e Neurocirurgia, Universidade Federal de São Paulo, São Paulo, SP, Brazil*
[f] *Neuroftalmologia, Chistian Business School, Orlando, Estados Unidos*
[g] *Centro Universitário Governador Ozanam Coelho, Ubá, MG, Brazil*
[h] *Centro Universitário Facisa - UNIFACISA, Campina Grande, PB, Brazil*
[i] *Medical Student, FIPMoc University Center, Montes Claros, Brazil*

ARTICLE INFO

Article history:
Received 21 June 2022
Accepted 27 June 2022

Keywords:
Wallenberg syndrome
Stroke
Neuro-ophthalmology

ABSTRACT

Wallenberg syndrome is also called lateral medullary syndrome, a neurological disorder resulting from occlusion of the vertebral artery or the posterior inferior cerebellar artery. The clinical presentations are associated with a variety of indications, including vestibulocerebellar symptoms, autonomic dysfunction and ipsilateral cerebellar signs. The ipsipulsion, an abnormality of the ocular movement associated with the Wallenberg syndrome, is more specific to the lateral medullary syndrome and is characterized by a tonic deviation of the eyes in the direction of the damaged side, more prominently when the visual fixation is interrupted. A 51-year-old male patient presented with a sudden permanent rotatory dizziness, unsteady gait, numbness in the left hemibody, left palate paresis, incoordination on left side and horizontal jerk nystagmus with left fast fase. Magnetic resonance imaging showed infarction in the left medulla and cerebellar. The ocular exam revealed saccadic lateropulsion ipsilateral to lesion. In the neurologic evaluation of the patient with Wallenberg syndrome, numerous abnormalities manifestations are present, such as vestibulo-ocular reflex

☆ Competing Interests: All authors declare no conflict of interest.

* *Corresponding author.*
E-mail address: cesar.claudino@unifesp.br (J.C. Claudino dos Santos).

https://doi.org/10.1016/j.radcr.2022.06.099

deficiency, saccadic abnormalities, low pursuance movements and gaze fixation, and eye alignment dysfunction. This semiologic feature had not been described in literature until now. We hypothesize that an initial vasogenic edema extending to the left medial medulla following the acute stroke could explain the early presentation with saccadic counterpulsion. After one week and regression of the edema, the finding of lateropulsion has alternated to the classic ipsipulsion related to Wallenberg syndrome. The following case report depicts a rare case of Wallenberg syndrome associated with alterations of the ocular motricity.

Introduction

The Wallenberg syndrome (WS), also known as lateral medullary syndrome, consists in an uncommon presentation of ischemic cerebrovascular accidents of the posterior circulation of the brain due to the occlusion of the intracranial segment of the vertebral artery followed by the posterior inferior cerebellar artery (PICA) and its branches [1–3]. This syndrome is characterized by vestibulocerebellar symptoms, autonomic dysfunction, sensorial symptoms and ipsilateral bulbar weakness [1,4]. Numerous abnormalities of the ocular movement are associated with the WS, including inclinational deviation, nystagmus horizontal/torsional/vertical, gaze evoked nystagmus and ipsipulsion [5,6]. The ipsipulsion is more specific to the lateral medullary syndrome and is characterized by a tonic deviation of the eyes in the direction of the damaged side, more prominently when the visual fixation is interrupted. In

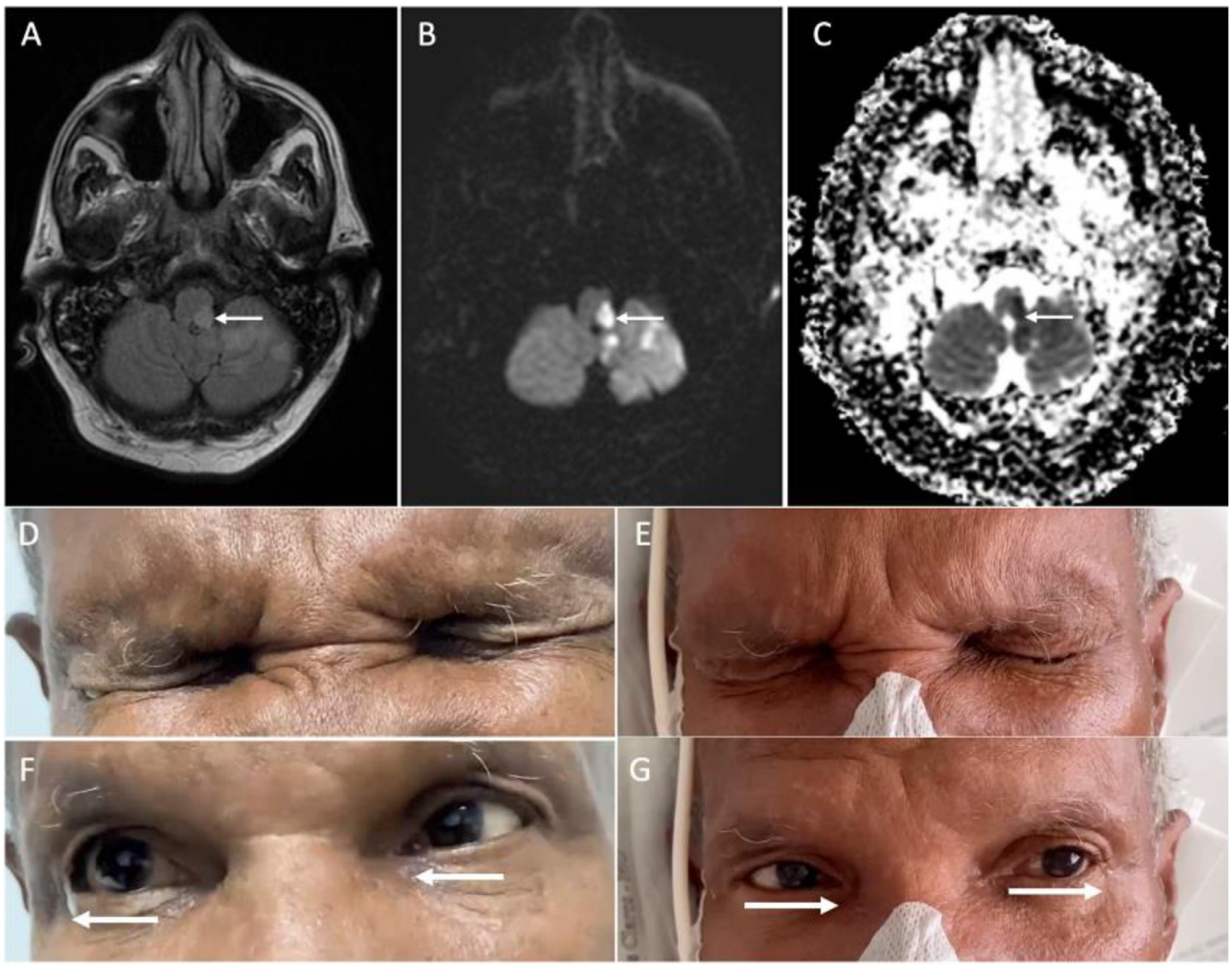

Fig. 1 – (A) Flair hyperintense focus at the left side of the medulla. (B) DWI sequences confirmed the ischemic event, (C) showing diffusion restriction. Classic features of ischemic infarct in left medulla. (D, F) Ocular contrapulsion followed by (E, G) ipsipulsion after 7 days.

contrast, in the lateropulsion, the voluntary saccades in the direction of the lesion are hypometric and the saccades in the opposite direction are hypermetric [7]. This case report aims to show a possible association between the ischemic cerebrovascular accident of the PICA and the ocular contrapulsacion followed by ocular ipsipulsion in the Wallenberg syndrome.

Case reports

A 51-year-old man with diabetes presented with a sudden permanent rotatory dizziness, unsteady gait and numbness in the left hemibody. He showed left palate paresis, incoordination on left side, horizontal jerk nystagmus with left fast fase and saccadic contrapulsion in ocular exam (Fig. 1) MRI showed left cerebellar hypodensity. MRI (Fig. 1) showed infarction in the left medulla and cerebellar. After 7 days of symptoms, he reported intermittent and frequent hiccups treated with chlorpromazine. The ocular exam at this time revealed saccadic lateropulsion ipsilateral to lesion (ipsipulsion) (Fig. 1) [8,9]. This semiologic feature had not been described in literature until now.

Discussion

A rare case of WS associated with alterations of the ocular motricity was described. The control of the gaze is situated diffusely in the central nervous system (CNS) in which the cerebellum and its pathways play a crucial role.

Oculomotor disturbance in WS

In the neurologic evaluation of the patient with Wallenberg syndrome, besides the symptoms of dissociated sensory damage, dysphagia, Horner syndrome and hemiataxia, neurological and ophthalmological manifestations are present, such as mild occulary deficits, including spontaneous nystagmus and gaze evoked nystagmus, as well as vestibulo-ocular reflex deficiency, saccadic abnormalities, low pursuance movements and gaze fixation, and eye alignment dysfunction [2,8,10,11].

Ocular contrapulsacion followed by ocular ipsipulsion

The cerebellum appears to be involved in the amplitude regulation of the saccadic eye movements [12]. It is acknowledged that the damage of the central cerebellar connections that go through the dorsolateral bulb is responsible for deficits of the ocular motricity [13]. Damages to the climbing, which carries information from the contralateral inferior olivary nucleus to the Purkinje cells in the oculomotomotor vermis ipsilaterally, which inhibits the fastigial nucleus and, finally, diminishes the excitatory saccadic stimulus for the complex paramedian pontine reticular formation-abducens nucleus in the opposite side, therefore resulting in a saccadic abnormality known as lateropulsion saccadic in the direction of the lesion [14,10]. The fixed gaze abnormalities in patients with Wallenberg syndrome can result in ipsipulsion of the ocular movements, noted after the patient closes the eyes or during a vertical saccadic movement (when we observe an elliptical trajectory of the eyes with the parable being ipsilesional). This fixed gaze disorder can also induce horizontal saccadic abnormalities. The lateral saccades away from the lesion side are hypometric, while the saccades directed to the lesion side are hypermetric. The ipsipulsion in patients with lateral medullary lesions is opposite to the saccadic counterpulsion that is associated with lesions of the medial medulla, with involvement of the climbing fibers before crossing the midline [11]. In the case of our patient, we hypothesize that an initial vasogenic edema extending to the left medial medulla following the acute stroke could explain the early presentation with saccadic counterpulsion. After one week and regression of the edema, the area of true infarction had probably become delimitated to the lateral medulla and, thereafter, the finding of lateropulsion has alternated to the classic ipsipulsion related to Wallenberg syndrome.

Conclusion

This report describes a rare oculomotor manifestation of the Wallenberg syndrome and brings a new vision about a possible topography possibly causing this clinical presentation.

Patient consent

Patient written and informed consent was obtained in order to write this article.

Acknowledgments

Medical School of University Center Christus, UNICHRISTUS, Fortaleza, CE, Brazil.

REFERENCES

[1] Ogawa K, Suzuki Y, Oishi M, Kamei S. Clinical study of 46 patients with lateral medullary infarction. J Stroke Cerebrovasc Dis 2015;24(5):1065–74.
[2] Lui F, Tadi P, Anilkumar AC. StatPearls, Treasure Island (FL): StatPearls Publishing; 2021. [Internet]Wallenberg syndrome.
[3] Kim JS. Pure lateral medullary infarction: clinical-radiological correlation of 130 acute, consecutive patients. Brain 2003;126(Pt 8):1864–72.
[4] Inamasu J, Nakae S, Kato Y, Hirose Y. Clinical characteristics of cerebellar infarction due to arterial dissection. Asian J Neurosurg 2018;13(4):995–1000.
[5] Waespe W, Wichmann W. Oculomotor disturbances during visual-vestibular interaction in Wallenberg's lateral medullary syndrome. Brain. 1990;113 (Pt 3):821-46.

[6] Hagström L, Hörnsten G, Silfverskiöld BP. Oculostatic and visual phenomena occurring in association with Wallenberg's syndrome. Acta Neurol Scand 1969;45(5):568–82.
[7] Meyer KT, Baloh RW, Krohel GB, Hepler RS. Ocular lateropulsion. A sign of lateral medullary disease. Arch Ophthalmol 1980;98:1614–16.
[8] Paliwal VK, Kumar S, Gupta DK, Neyaz Z. Ipsipulsion: a forgotten sign of lateral medullary syndrome. Ann Indian Acad Neurol 2015;18(3):284–5.
[9] Kim JS, Moon SY, Park SH, Yoon BW, Roh JK. Ocular lateropulsion in Wallenberg syndrome. Neurology 2004;62(12):2287.
[10] LeighRJ Zee DS. The neurology of eye movements. Philadelphia, FA: Davis Company; 1991. p. 423–4.
[11] Uno A, Mukuno K, Sekiya H, Ishikawa S, Suzuki S, Hata T. Lateropulsion in Wallenberg's syndrome and contrapulsion in the proximal type of the superior cerebellar artery syndrome. Neuro Ophthalmol 1989;9:75–80.
[12] Keller EL. The cerebellum. In: Wurtz RH, Goldberg ME, editors. The neurobiology of saccadic eye movements. Amsterdam: Elsevier; 1989. p. 391–411.
[13] Waespe W, Wichmann W: 2022 Oculomotor disturbances during visual-vestibular interaction in Wallenberg.
[14] Kommerell G, Hoyt WF. Lateropulsion of saccadic eye movements: electro-oculographic studies in a patient with Wallenberg's syndrome. Arch Neurol 1973;28:313–18.