Case Report

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

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A B S T R A C T

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, incordination on left side and horizontal jerk nystagmus with left fast phase. 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.
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.

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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 oculomot-
omic syndrome, 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 palpe paresis, incoordi-
nation on left side, horizontal jerk nystagmus with left fast
saccades and saccadic contrapulsion in ocular exam (Fig. 1) MRI
 showed left cerebellar hypodensity. MRI (Fig. 1) showed infar-
tion in the left medulla and cerebellar. After 7 days of symp-
toms, 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 litera-
ture 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 dam-
age, dysphagia, Horner syndrome and hemiataxia, neurolog-
ical and ophthalmological manifestations are present, such as
mild oculary 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 contrapulsion following by ocular ipsipulsion

The cerebellum appears to be involved in the amplitude regu-
lation 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 oculomotor vermis ipsilater-
ally, which inhibits the fastigial nucleus and, finally, dimin-
ishes the excitatory saccadic stimulus for the complex para-
median 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 Wallen-
berg syndrome can result in ipsipulsion of the oculomove-
ments, noted after the patient closes the eyes or during a ver-
tical saccadic movement (when we observe an elliptical trajec-
tory of the eyes with the parable being ipsilesional). This fixed
gaze disorder can also induce horizontal saccadic abnormali-
ties. The lateral saccades away from the lesion side are hypo-
metric, while the saccades directed to the lesion side are hy-
permetric. The ipsipulsion in patients with lateral medullary
lesions is opposite to the saccadic counterpulsion that is as-
associated with lesions of the medial medulla, with involve-
ment of the climbing fibers before crossing the midline [11].
In the case of our patient, we hypothesize that an initial va-
sogenic edema extending to the left medial medulla follow-
ing 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 de-
limited 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 possi-
tble topography possibly causing this clinical presentation.

Patient consent

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

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