

# A case of double depressor palsy followed by pursuit deficit due to sequential infarction in bilateral thalamus and right medial superior temporal area

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## Abstract

**Background** We present a unique case of a patient who suffered two rare events affecting the supranuclear control, first of the vertical and second of the horizontal eye movements. The first event involved bilateral thalamic infarcts that resulted in double depressor palsy. The second event occurred 1 year later and it involved supranuclear control of horizontal eye movements creating pursuit deficit.

**Case presentation** A 47-year-old male presented with complaints of diplopia upon awakening. He had atrial fibrillation, mitral valve regurgitation, aortic valve regurgitation, and a history of spleen infarction

1 year ago. His right eye was hypertrophic and right eye downgaze was limited unilaterally of equal degree in adduction and abduction. The patient was diagnosed with double depressor palsy of the right eye. Magnetic resonance imaging (MRI) of the brain showed an old infarction of the left thalamus, and diffusion MRI showed acute infarction of the right thalamus. The patient's daily warfarin dose was 2 mg and it was increased to 5 mg with cilostazol 75 mg twice a day. Seven weeks later, the patient's ocular movement revealed near normal muscle action, and subjectively, the patient was diplopia free. At follow-up 12 months later, the patient revisited the hospital because of sudden onset of blurred vision on right gaze. He was observed to have smooth pursuit deficit to the right side, and orthophoric position of the eyes in primary gaze. MRI of the brain showed an acute infarction in the right medial superior temporal area.

**Conclusions** The patient experienced very rare abnormal eyeball movements twice. This case highlights the importance of evaluating vertical movement of the eyes and vascular supplies when patients present with depressor deficit and supports the theory of a supranuclear function in patients who present with pursuit deficit.

**Keywords** Artery of Percheron · Bilateral thalamic infarction · Double depressor palsy · Medial superior temporal area · MST · Monocular depression palsy · Parieto-temporal-occipital junction infarct · Pursuit deficit

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## Abbreviations

MRI	Magnetic resonance imaging
MST	Medial superior temporal area
MDD	Monocular depression deficiency
riMLF	Rostral interstitial nucleus of the medial longitudinal fasciculus
VOR	Vestibulo–ocular reflex
OKN	Optokinetic nystagmus
IR	Inferior rectus
SR	Superior rectus
CT	Computed tomography
CN	Cranial nerve
INC	Interstitial nucleus of Cajal
AOP	Artery of Percheron
MT	Medial temporal visual area
DLPN	Dorsolateral pontine nuclei
NRTP	Nucleus reticularis tegmenti pontis

## Background

Double depressor palsy is a condition characterized by deficiency of depression of one eye (unilateral limitation of downgaze), and it is also called monocular depression deficiency [1]. It is mainly congenital, but it can be caused by acquired primary inferior rectus palsy, inferior rectus paralysis due to supranuclear lesions, and primary or secondary contracture of the superior rectus. However, the definite cause has not yet been established [1, 2].

Muscles that are involved in depression are the inferior rectus and superior oblique; these two muscles are innervated by two different ocular motor nuclei, ipsilateral the oculomotor nucleus, and contralateral the trochlear nucleus. Therefore, the oculomotor nucleus on one side and the trochlear nucleus on the other side are usually not affected simultaneously. The supranuclear pathway for downgaze is bilaterally represented in the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF) located in the midbrain. Only bilateral lesions of the riMLF cause downgaze paresis. Isolated downward gaze paralysis in one eye that is present in both abduction and adduction is unique because of the neurologic supply. The depressors in one eye include the inferior rectus (supplied by ipsilateral medial riMLF) and superior oblique (supplied by contralateral medial riMLF). It is unlikely that one lesion would affect both ipsilateral and contralateral medial riMLF. Two small focal

lesions in that region would be necessary, which makes this case unique. In contrast, bilateral downgaze palsy can occur from one single large unilateral lesion in the region of the riMLF affecting downgaze in both eyes [1–7]. Because bilateral lesions are much less common than unilateral lesions, downgaze disorders are much less common than those affecting upgaze. The reports presenting cases of double depressor palsy are rare [4–8].

We report a case of a patient with downgaze paralysis, in whom magnetic resonance imaging (MRI) revealed discrete bilateral lesions that included left paramedian thalamic infarction and right new paramedian thalamic infarction.

Medial superior temporal area (MST) is located at the parieto–temporal–occipital junction process visual motion signals that drive pursuit. The cerebral cortex primarily controls ipsiversive pursuit and hemispheric lesions generally impair pursuit toward the side of the lesion [9]. A unilateral lesion affecting area MST results in a decrease in smooth pursuit gain, bilaterally but more markedly in the movement directed ipsilaterally to the lesion [10]. The asymmetrical pursuit deficit can lead to a misdiagnosis of a pseudo-abducens nerve palsy. It could be ruled out by checking for optokinetic nystagmus (OKN) drum [11].

This patient experienced two rare events affecting the supranuclear control first of the vertical and secondly of the horizontal eye movements. The first event was double depressor palsy due to bilateral thalamic infarcts. The second event occurred one year later and it involved supranuclear control of horizontal eye movements creating pursuit deficit.

## Methods and results

A 47-year-old man with atrial fibrillation awoke with double vision. His mental status was alert and neurologic examination was normal. His medical history was notable for mitral valve regurgitation and aortic valve regurgitation as well as atrial fibrillation, which had resulted in previous splenic infarction. The diplopia was stationary after onset, aggravated on downward gaze, and not affected by posture. He had a subtle chin down posture and complained difficulty in reading, but had not head tilt posture. The type of diplopia was mainly vertical, but horizontal or torsional diplopia was not perceived. The eye signs were as follows: Best-

corrected visual acuities were 20/25 + 1 in the right eye (OD) and 20/25 + 2 in the left (OS), the intraocular pressure was 15 mmHg in both eyes and visual fields were full. The pupils were reactive to light and both pupils were 4 mm. Convergence was in the normal range and nystagmus was not found in all gaze positions. Lids were normal and retraction was not detected. At rest, the right eye was deviated upward and was exotropic, distance cover–uncover test revealed 14 prism diopters of right hypertropia and two prism diopters of OD exotropia in primary gaze. Downward gaze was limited in primary position, adduction, and abduction. The right hypertropia was 18 prism diopters in right and down, 16 prism diopters in left and down. The eye movements of the left eye were normal (Fig. 1). On funduscopic exam, there was no obvious cyclotorsion in either eye. The binocular vision test findings were as follows: Worth-4-dot test revealed vertical diplopia in primary position; titmus and randot stereopsis were normal. On Maddox rod testing, vertical deviation of right eye was found, but cyclodeviation was not found. Downgaze paralysis of the right eye was found on the Hess screen test, the amount of attempted depression did not change on adduction or abduction (Fig. 2). Vestibulo–ocular reflex (VOR), demonstrated by the doll’s head maneuver, was intact. On force generation test, muscle power of the right eye was weak on downgaze and muscle was not restricted on the force duction test. His initial laboratory tests showed white blood cell count, 7400/ml; red blood cell count,  $4.75 \times 10^6/\mu\text{l}$ , hemoglobin, 15.8 g/dl; platelets,  $202 \times 10^3$ ; blood urea nitrogen, 15.5 mg/dl; creatine, 1.1 mg/dl. ESR, CRP, CK-MB, TnT, and proBNP were in normal range. Thyroid function tests showed TSH, 2.16  $\mu\text{UI/ml}$ ; total T3 0.82 ng/ml; free T4, 1.10 ng/dl. Anti-Ach-R Ab was negative and PT/INR was 16.9/1.5. Neurologic exam was performed by

neurologist; mental status, sensation (light touch), motor (arm drift, finger tap, hip flexors, and ankle dorsiflexors), coordination (finger to nose and heel to shin), gait, and deep tendon reflex test. These neurologic screening tests did not show any abnormal findings.

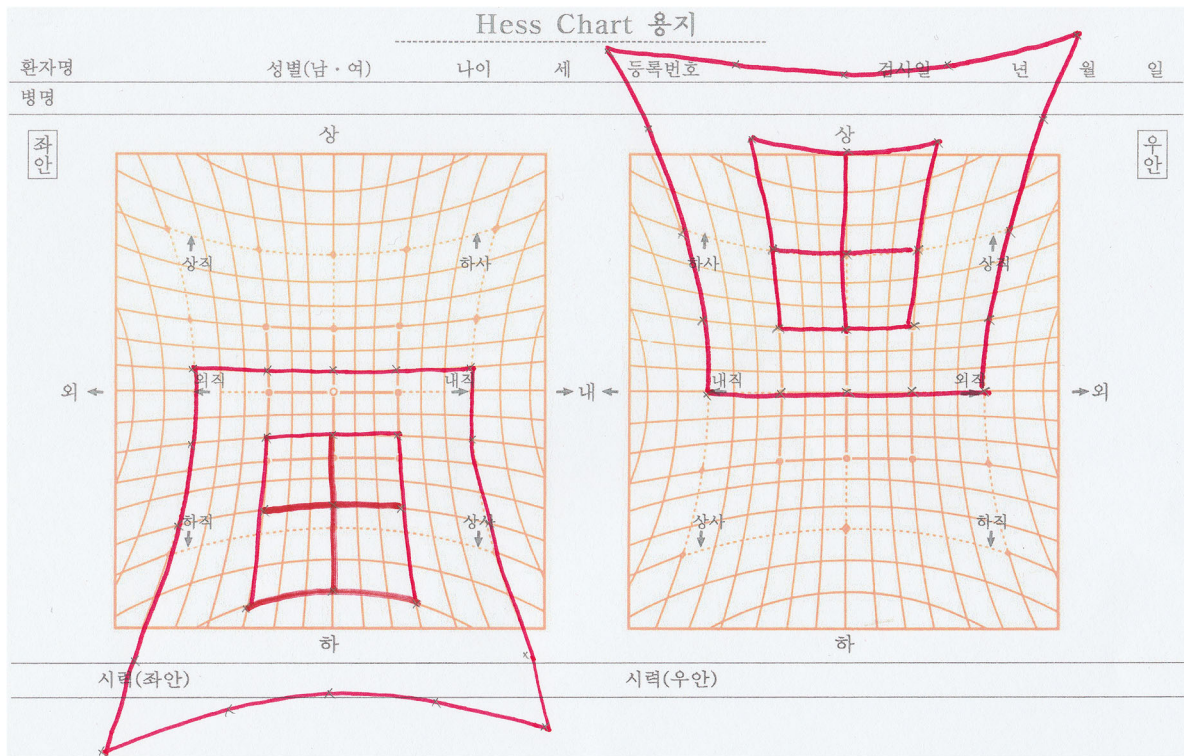
Magnetic resonance imaging and magnetic resonance angiography of the brain showed T1-weighted hypointense signal and T2-weighted hyperintense signal over the left paramedian thalamus and diffusion-weighted hyperintense signal over the right paramedian thalamus (Fig. 3). There was no cerebral aneurysm or vascular malformation.

The patient was diagnosed with old left paramedian thalamic infarction and acute right paramedian thalamic infarction. The patient’s daily warfarin sodium (Dae hwa Pharmaceutical Co., Gangwon-do, Korea) dose was 2 mg, and it was increased to 5 mg with cilostazol (Otzuka Pharmaceutical Co., Tokyo, Japan) 75 mg twice a day by neurologist. We prescribed add-on (Fresnel) prisms as 14 prism diopters base down on spectacles of the right eye to improve the diplopia and reestablish fusion, but the patient was not satisfied with the Fresnel prism because of the cosmetic issue. So we recommended six prism diopters base-down prism incorporated into the glasses of the right eye. As the result, the subjective diplopia and reading difficulty were improved with the prism. Seven weeks later, hypertropia of the right eye was improved in the primary and six cardinal position, and limitation of downgaze had subsided completely, and subjectively, the patient was diplopia free.

At follow-up 12 months later, the patient revisited the hospital because of sudden onset of blurred vision on right gaze. The patient could not distinguish the horizontal diplopia and blurred vision exactly, but the symptom had been observed for 3 days. Alternate



**Fig. 1** In primary position, hypertropia of the right eye was found, and limitation to downgaze of equal degree in adduction and abduction was noted



**Fig. 2** Hess charting was consistent with palsy of the superior oblique and inferior rectus of the right eye

prism cover test revealed six prism diopters of OD esotropia in right gaze, but orthophoric in primary gaze, other cardinal and tertiary gaze. Convergence was intact. The patient did not follow the target moving to the right side, and the pursuit deficit to the right side was observed with the OKN drum.

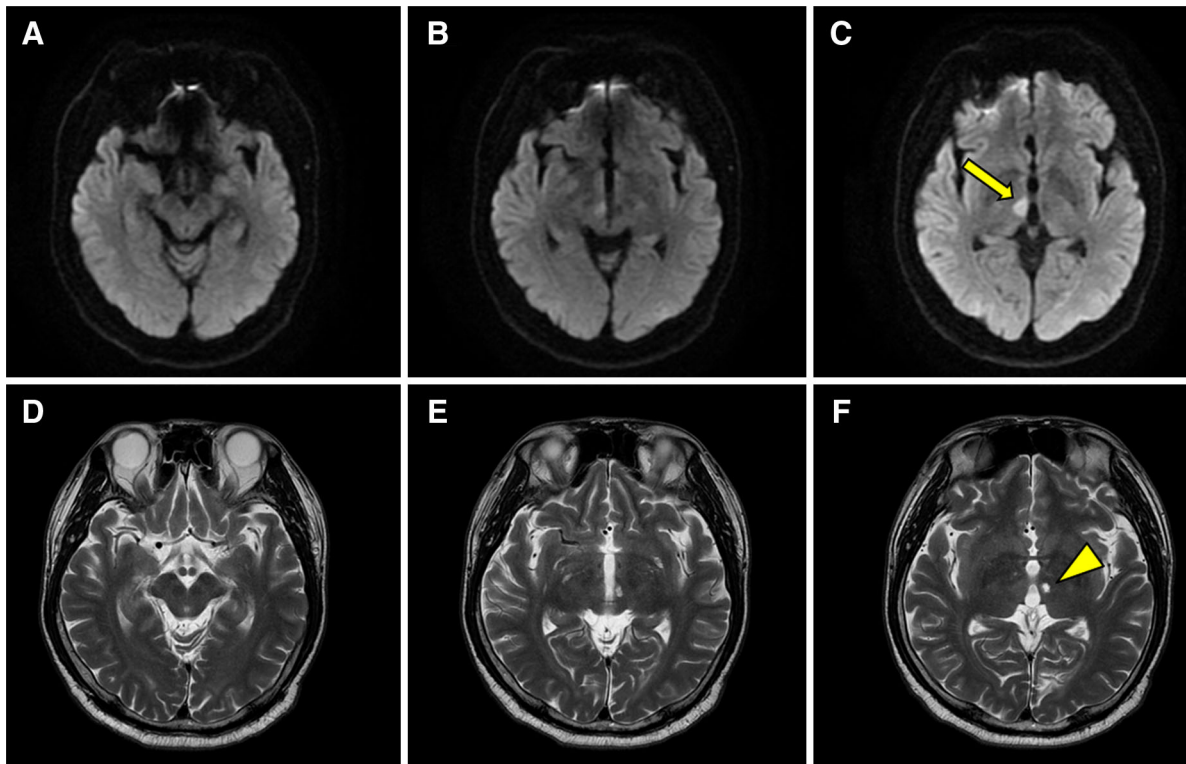
On ocular examination, the patient's best-corrected visual acuities were 20/25 + 1 in the right eye (OD) and 20/25 + 2 in the left (OS). Pupils were equal, round, and reactive to light, and there was no evidence of afferent pupillary defect or miosis. Visual field test was normal. No cyclodeviation was found on double Maddox rod testing, and no vertical deviation was found upon single Maddox rod testing. VOR, demonstrated by the doll's head maneuver, was intact, allowing full abduction of the right eye. No apparent limitation of both eyes was found on the Hess screen test, and no marked diplopia was found on the red glass test. Cranial nerve testing, excluding extraocular motility, was normal. MRI of the brain showed a focal diffusion restricted lesion in the right occipital lobe which was diagnosed as acute infarction (Fig. 4).

## Discussion

### Event #1: the vertical gaze deficit

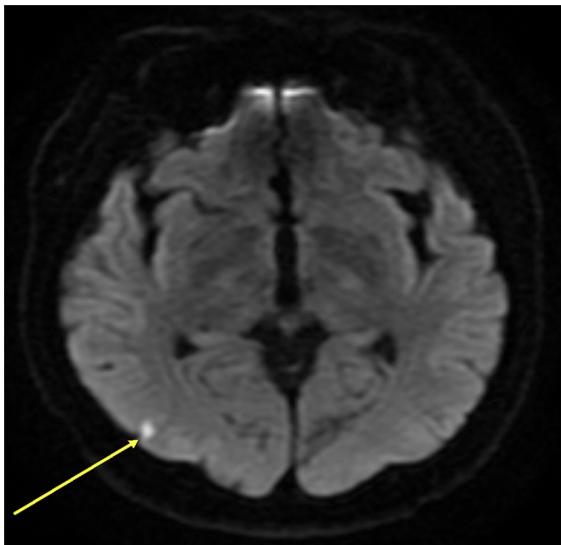
Acquired double depressor palsy is usually caused by cerebrovascular disease affecting the pons or cerebellum. The initial presenting symptoms depend on the site and extent of the lesion, but patients usually present with impaired consciousness [1].

Double depressor palsy is characterized by unilateral downgaze deficiency of equal degree in adduction and abduction. The affected eye is hypertrophic and may cause over-elevation in adduction and abduction. When the unaffected eye fixes, the paretic eye may have upper eye lid retraction. The lid is drawn back passively when the globe is hypertrophic because of the fascial attachments between the SR and levator palpebrae. Pseudoptosis may occur on attempted downgaze when the paretic eye does not depress, but the lids lower bilaterally, and pseudoptosis of the non-involved eye may also occur when patient fixates with involved eye (right eye) in primary gaze. Also, binocularity could worsen because of amblyopia or



**Fig. 3** Axial view of brain MRI. **a–c** Diffusion MRI images show high signal intensity in the right paramedian thalamus (*arrow*); acute infarction. Midbrain is intact. **d–f** T2-weighted

images show high signal intensity in the left paramedian thalamus (*arrow head*); old infarction. Midbrain is intact



**Fig. 4** Axial view of brain MRI: diffusion MRI image shows high signal intensity in the right parieto-temporal-occipital junction (*arrow*); acute infarction

suppression when double depressor palsy remains for a long time in young children. Convergence may be impaired when prolonged comitant hypertropia inhibits convergence [1, 2].

Vestibulo-ocular reflexes (doll's head maneuvers) are usually intact on downgaze, indicating a supranuclear etiology with preservation of the nuclear reflexes. When a patient with conjugate horizontal, vertical, or complete ophthalmoparesis is encountered, it is important to determine if the lesion is supranuclear (upper motor neuron) or nuclear-infranuclear (lower motor neuron). Supranuclear lesions affect the saccadic, smooth pursuit, optokinetic, or vergence inputs in the brainstem, whereas nuclear-infranuclear lesions affect ocular motor nuclei, nerves, neuromuscular junction, or extraocular muscles themselves. The distinction can be made at the bedside by checking if the ophthalmoparesis can be overcome with the VOR, as elicited with head movements (i.e., the doll's eyes or oculocephalic maneuver) or caloric stimulation [12].

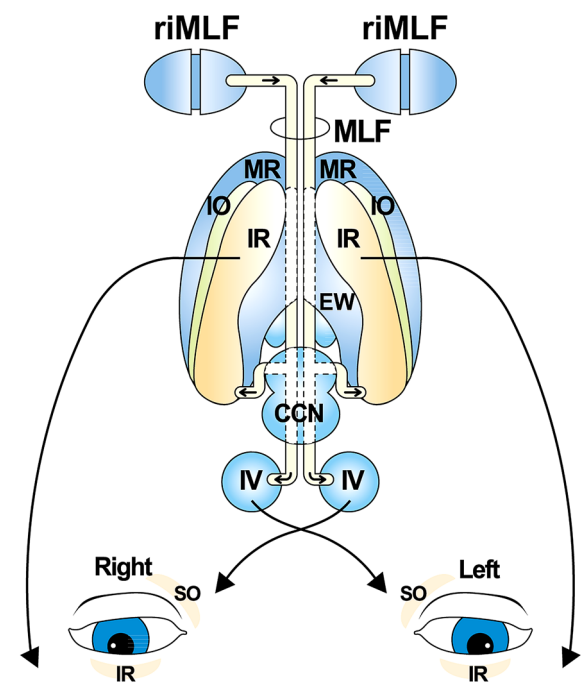
Acquired disorders that can mimic double depressor palsy include a traumatic orbital blow-out fracture with associated IR palsy or entrapment and thyroid orbitopathy with SR contracture, skew deviation, myasthenia gravis, and oculomotor nerve palsy. Previous strabismus surgery with excessive recession of the IR, a large resection of the SR, or scar tissue formation may cause diminished downgaze.

Skew deviation should be considered in the differential diagnosis. Eyeball movements are not limited in skew deviation, vertical diplopia may be comitant or incomitant, and it can subside in the supine position. Also, it is associated with ocular torsion and head tilt, incyclotorsion in the hypertrophic eye, and excyclotorsion in the hypotrophic eye. Skew deviation is accompanied by neurologic symptoms related to brainstem or cerebellum [1, 2]. Our patient presented with unilateral downgaze deficiency of similar degree in adduction and abduction and had incomitant diplopia. But he did not have a head tilt and ocular torsion which could indicate skew deviation or any other symptoms of the brainstem or cerebellum; therefore, skew deviation can be ruled out. He did not have any other signs of ocular tilt reaction.

Myasthenia gravis should be also considered in the differential diagnosis, because any muscle or combination of muscles may be affected, and myasthenia may mimic any disorder of eye movement. Ptosis and underaction of extraocular muscles are common variables, and if the patient has a ptosis with abnormal deviation, or there is a history of variability through the day, with symptom worsening when the patient is tired, or horizontal deviation is accompanied by vertical deviation or ocular movement is limited in the version or duction test and the symptoms are variable during the follow-up period, the diagnosis of myasthenia gravis can be assumed clinically [2].

The supranuclear brainstem center for control of conjugate vertical and torsional eye movements is the riMLF, which is located in the midbrain. The riMLF is located dorsomedial to the rostral end of the red nucleus, lateral to the periaqueductal gray matter, superior to the interstitial nucleus of Cajal (INC) [13]. The riMLF contains saccadic burst neurons and receives an inhibitory input from omnipause neurons in the nucleus raphe interpositus. Excitatory burst neurons in the riMLF project to neurons of cranial nerve (CN) III and CN IV, and send an axon collateral to the INC. Inhibitory burst neurons are located in the INC [13, 14].

The excitatory neurons that control depressor muscles (IR, SO) are located rostrally, and the neurons that control elevator muscles (SR, IO) are located caudally. The burst neurons of medial (rostral) riMLF project to the ipsilateral oculomotor nucleus (to ipsilateral IR) and the ipsilateral trochlear nucleus (to contralateral SO), and the neurons of lateral (caudal) riMLF project to the contralateral oculomotor nucleus (to contralateral IO and ipsilateral superior rectus) (Fig. 5). Therefore, only bilateral lesions cause double depressor palsy. The INC which is the neural integrator of vertical and torsional gaze receives signals from the riMLF and from the vestibular nuclei and projects to the motoneurons of the CN III and CN IV nuclei through the posterior commissure (PC). Information involved in upgaze crosses in the PC. Excitatory signals of INC, which is stimulated from riMLF, cross the posterior commissure and inhibit contralateral neurons of the INC involved in downgaze. Information related to downgaze inhibits



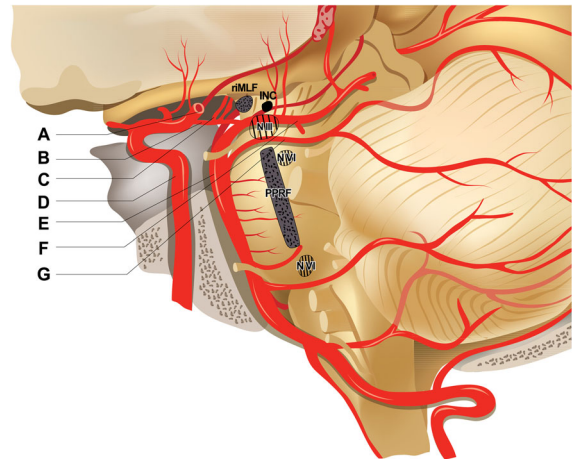
**Fig. 5** The excitatory neurons that control depressor muscles (IR, SO) are located rostrally, and the neurons that control elevator muscles (SR, IO) are located caudally. The burst neurons of medial (rostral) riMLF project to the ipsilateral oculomotor nucleus (to ipsilateral IR) and the ipsilateral trochlear nucleus (to contralateral SO), and the neurons of lateral (caudal) riMLF project to the contralateral oculomotor nucleus (to contralateral IO and ipsilateral superior rectus)

neurons of the INC related to upgaze without decussation. Unilateral or bilateral lesions of the riMLF usually result in conjugate gaze palsy [1–6, 13–15]. The riMLF receives afferent fibers from the frontal eye field (Brodmann area 8) and the superior vestibular nucleus, and links to paramedian reticular formation [9].

The blood supply to the riMLF is via a branch of the basilar communicating artery, namely the posterior thalamic paramedian artery [1, 3]. It is called the posterior thalamoperforating artery and the main perforating trunk from P1 (posterior cerebral artery) is called a thalamoperforating artery [1, 16–21]. The thalamoperforating arteries were identified by both Heubner [22] and Duret [23] in the latter part of the nineteenth century, and they were described as “optic arteries” supplying part of the thalamus and the optic pathways. The thalamoperforating artery arises from the posterior cerebral artery or posterior communicating artery. The basilar communicating artery emanates from the top of the basilar artery, and is sometimes called the P1 segment of the posterior cerebral artery (Fig. 6).

The thalamoperforating arteries arising from the posterior communicating artery are called the anterior thalamoperforating arteries (Fig. 7d), and the thalamoperforating arteries arising from the posterior cerebral artery are named the posterior thalamoperforating arteries [19, 23] (Fig. 7a–c). The thalamoperforating arteries are divided to three segments: interpeduncular segment, mesencephalic segment, and thalamic segment. The thalamoperforating arteries penetrate the floor of the third ventricle to supply the subthalamic region including the riMLF, medial rostral red nucleus, posteroinferior dorsomedial nucleus of the thalamus, nucleus parafascicularis, and medial portion of the centromedian nucleus of the thalamus [4, 16–20].

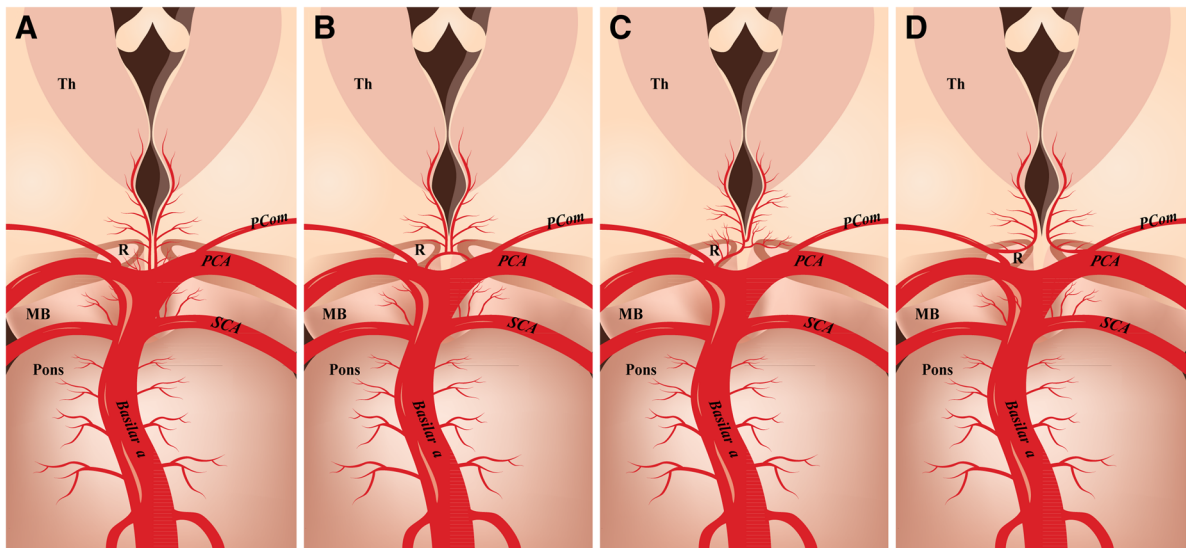
In this case, two separate episodes of thalamoperforating artery infarction occurred. The paramedian arteries (thalamoperforating arteries) have great variability with respect to number, size, and territorial contribution to the thalamus [16]. Percheron described three different patterns for the origin of thalamic paramedian arteries. In type I, a pair of thalamic paramedian arteries is present, each as a branch of the respective basilar communicating artery. This is the most common form, in which bilateral small perforating arteries arise from the P1 segments of the



**Fig. 6** A Middle cerebral artery, B posterior communicating artery, C thalamoperforating artery, D left internal carotid artery, E Basilar artery, F posterior cerebral artery, G superior cerebellar artery. The blood supply to the riMLF is via a branch of the basilar communicating artery, namely the posterior thalamic paramedian artery. It is called the posterior thalamoperforating artery and the main perforating trunk from P1 (posterior cerebral artery) is called a thalamoperforating artery

posterior cerebral arteries (Fig. 7a). In type II, both thalamic paramedian arteries arise from a single basilar communicating artery, frequently with a common stem (Fig. 7c). Type III was described as an arcade of perforating arteries arising from an artery bridging the bilateral P1 segments and it is a less common pattern (Fig. 7b). The type II pattern was identified in approximately half of Percheron’s specimens, and it obviously predisposed to bilateral lesions resulting from single vessel occlusion [24]. The type II variation is called the artery of Percheron (AOP). The AOP is an uncommon anatomic variant, in which a single dominant thalamoperforating artery supplies the bilateral medial thalami with variable contribution to the rostral midbrain. Occlusion results in a characteristic pattern of ischemia: bilateral paramedian thalamic infarcts with or without midbrain involvement. Rodriguez et al. [26] described subacute infarcts in the bilateral paramedian thalami from a single common trunk arising from the left P1 segment with double-branching vessels distally. Their diagnosis of occlusion of the vascular variant, AOP, was made. Pal [27] reported a case of double depressor palsy caused by bilateral paramedian thalamic infarcts without midbrain lesion, which resulted from AOP occlusion.

In this case, two separate events of infarction were presented: a left old paramedian infarct and a right



**Fig. 7** The thalamoperforating arteries, schematic representation, coronal plane-posterior third ventricle level. The posterior thalamoperforating artery (a–c). **a** The most common variation, there are many small perforating arteries arising from the P1 segments of the PCA. (P1 segment: segment from the basilar artery to the junction with the PCom.) **b** An arcade of perforating

branches arising from the PCA. **c** The artery of Percheron is a single perforating blood vessel arising from one P1 segment. **d** The anterior thalamoperforating arteries arising from the Pcom arteries. *PCom* a posterior communicating artery, *PCA* posterior cerebral artery, *MB* midbrain, *Th* thalamus, *B* basilar artery, *R* red nucleus, *S* substantia nigra, *P* basis pedunculi

new paramedian infarct. So the patient is assumed to have a pair of thalamic paramedian arteries arising from the respective basilar communicating artery, not the AOP (Fig. 7a or d).

The polar artery is absent in 30–60% of the population [13], and thalamoperforating arteries can supply both the paramedian and the anterior thalamic territories, especially when the polar artery is absent. Additionally, the arteries that supply the rostral midbrain—the superior mesencephalic (or rubral) arteries—can branch separately from P1 or share a common origin with the paramedian arteries [16–20].

Massimo et al. [3] reported eight cases of bilateral paramedian thalamic artery infarcts, and their main symptoms were disorder of vigilance and hypersomnolence, amnesia, changes of mood and bulimia, and vertical gaze paresis. Our patient presented with only vertical diplopia without other neurologic symptoms, and there was no neurologic deficit except for double depressor paresis.

The common cause of double depressor palsy is bilateral infarctions of the riMLF, and bilateral midbrain infarcts which result from one event of the AOP (Fig. 7c) are representative [1, 25, 27]. Double depressor palsy is also caused by a periaqueductal

lesion [28], which is probably because of involvement of afferent downgaze fibers projecting to the riMLF. Other possible sites affecting downgaze include the interstitial nucleus of Cajal [1] and the posterior commissure [1, 6]. However, the mechanisms by which bilateral riMLF lesions result in only one-sided double depressor palsy and the significance of bilateral lesions as opposed to unilateral lesions have remained controversial [4].

In our patient, T1- and T2-weighted MR imaging revealed a left old paramedian infarct and diffusion-weighted MR imaging demonstrated a right new paramedian infarct. Our patient had high-risk factors for cerebrovascular accident, and when the left paramedian thalamic infarct occurred, he did not notice the event and did not have diplopia at all. The superior oblique in right eye (supplied by left medial riMLF) could have been weakened, but the inferior rectus in right eye (supplied by right riMLF), the other depressor had maintained its function at that time. This can explain why the patient had not had any vertical diplopia and reading difficulty. After new infarct occurred in right paramedian thalamus resulting from right thalamoperforating artery occlusion, he had vertical diplopia because the remained depressor



of right eye, the inferior rectus was affected. Therefore, this case indicates that only bilateral lesions in medial riMLF would be able to cause the double depressor palsy.

In our patient, MRI demonstrated bilateral paramedian thalamic infarcts without midbrain lesions because it is difficult to reveal the occlusion of small vessels supplying the riMLF on imaging. The arteries supplying the paramedian thalami also supply the midbrain including the riMLF, and although midbrain lesions were not detected, we could assume the deficit in bilateral riMLF.

We cannot rule out the possibility that double depressor palsy may result from bilateral paramedian thalamic infarcts without actual midbrain infarcts. Also, riMLF is located in the midbrain just rostral to the oculomotor nucleus at the junction of the midbrain and thalamus. We need to perform further study for assessing the supranuclear mechanisms of vertical eyeball movements.

Pal [27] reported a case of double depressor palsy caused by bilateral paramedian thalamic infarcts without midbrain lesion, which resulted from AOP occlusion. Contrary to previous reports, our patient had two separate cerebrovascular events; in other words, sequential bilateral paramedian infarcts without midbrain lesions resulted in double depressor palsy.

#### Event #2: the horizontal gaze deficit

The patient later presented with acute horizontal gaze limitation of voluntary eye movements that could be overcome by stimulation of the VOR. In this report, the lesion of the occipital lobe was corresponded to the medial temporal area (MST). MST area is located at the parieto-temporal-occipital junction process visual motion signals that drive pursuit. The cerebral cortex primarily controls ipsiversive pursuit and hemispheric lesions generally impair pursuit toward the side of the lesion [9].

The smooth pursuit system for visual targets originates in V5—the human homologue of the medial temporal (MT) visual area in the rhesus monkey—where it receives input from the primary visual system, both from the cortex (striate and extrastriate areas) and likely from magnocellular input directly from the geniculate. The MST area is also involved in

generating pursuit signals in response to moving stimuli. The area appears to be supplied with information about head movement as well as eye movement commands, and thus is critical in generating pursuit movements to follow a target while the head is moving. Information from the MT and MST projects via the posterior portion of the internal capsule to the dorsolateral pontine nuclei and lateral pontine nuclei, including the nucleus reticularis tegmenti pontis. From these pontine nuclei, projections are sent to the cerebellum [29].

A unilateral lesion affecting area MST results in a decrease in smooth pursuit gain, bilaterally but more markedly in the movement directed ipsilaterally to the lesion [29]. The pursuit deficit is to the side of the lesion. To test the pursuit system, the examiner should ask the patient to track a small target, such as a pencil tip held a meter or more from the eyes, with the head held still. The target should initially be moved at a low, uniform velocity. Pursuit movements that do not match the target velocity result in corrective saccades. If these are “catch-up” saccades, then the pursuit gain is low. And handheld drums or tapes with repetitive targets stimulate pursuit and may be useful in the detection of pursuit asymmetries [11].

The asymmetrical pursuit deficit can mislead to diagnosis of a pseudo-abducens nerve palsy. Pseudo-abducens palsy was defined as a failure of ocular abduction which is not due to dysfunction of the sixth nerve. This rare condition can be observed when voluntary eye movements show impairment in lateral gaze, yet the VOR demonstrates full abduction. The intact VOR indicates the integrity of the infranuclear abducens nerve, thus suggesting that a pseudo-abducens palsy is likely to be caused by supranuclear pathology [30]. The anatomical mechanism to explain the clinical presentation of pseudo-abducens palsy has been debated. The most compelling evidence in the literature regarding causative pathology is focused on supranuclear pathways in the midbrain, rostral pons, and thalamus [30, 31]. Wiest et al. [32] demonstrated esotropia and pseudo-abducens palsy in a patient with MST evidence of symmetric paramedian thalamic infarction, in the absence of midbrain pathology.

This case showed pursuit deficit in the movement directed ipsilaterally to the lesion of right parieto-temporal-occipital junction.

## Conclusions

The patient experienced very rare abnormal eyeball movements twice. The first event involved bilateral thalamic infarcts that resulted in double depressor palsy. The second event occurred 1 year later, and it involved supranuclear control of horizontal eye movements creating pursuit deficit. This case highlights the importance of evaluating vertical movement of the eyes and vascular supplies when patients present with depressor deficit and supports the theory of a supranuclear function in patients who present with pursuit deficit.

**Authors' contributions** KS is responsible for acquisition of the clinical information and writing up of the manuscript. YM is responsible for acquisition of the clinical information and reviewing the manuscript. LS is responsible for contributing to and reviewing the manuscript. All authors read and approved the final manuscript.

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## Compliance with ethical standards

**Conflict of interest** The authors K.S., L.S., and Y.M. declare that they have no conflict of interest.

**Informed consent** Informed consent was obtained from the participant included in the study.

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