Case Report

A rare case of medial medullary syndrome with upbeat nystagmus due to protein 'S' deficiency and hypothyroidism (Hashimato's thyroiditis) - A case report

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Abstract

Brainstem strokes frequently present challenges to diagnosis from both clinical and radiological stand points. The medulla has a compact arrangement of structures, and hence a single lesion usually gives a constellation of signs that constitute a syndrome. There are two basic ischemic vascular syndromes of the medulla: the more common lateral medullary infarction and the rare medial medullary infarction. The most classical presentation of medial medullary stroke is Dejerine syndrome, which consists of the triad of ipsilateral hypoglossal palsy, contralateral hemiparesis and contralateral lemniscal (spinothalamic) sensory impairment. The most common eye movement abnormality is upbeat nystagmus. In this case report we present a patient who developed Rt. Medial Medullary infarction, due to thrombosis of vertebral or anterior spinal artery, secondary to Protein 'S' Deficiency along with hypothyroidism.

Key words

Medial Medullary Syndrome, Upbeat Nystagmus, Protein 'S' Deficiency, Hypothyroidism, Hashimato's Thyroiditis.

Introduction

The medulla has a compact arrangement of structures, and hence a single lesion usually gives a constellation of signs that constitute a syndrome. There are two basic ischemic vascular syndromes of the medulla: the more common

lateral medullary infarction and the rare medial medullary infarction (MMI). The other even less common medullary syndromes, namely bilateral medial medullary, bilateral lateral medullary, and hemimedullary infarctions, are combinations of these two basic patterns. Furthermore, each of these syndromes may be "complete" or "incomplete" [1]. Medial medullary infarction (MMI) syndrome was initially described by Spiller more than 100 years ago [2], and Dejerine proposed a triad of symptoms: contralateral hemiplegia sparing the face, contralateral loss of deep sensation, and ipsilateral hypoglossal paralysis [3].

Pathological examination first conducted in 1937 demonstrated thrombotic occlusion of the anterior spinal artery (ASA) and adjacent vertebral artery (VA) [4]. More recently, studies using MRI have rapidly expanded our understanding of MMI syndromes [5, 6, 7, 8, 9].

Medial medullary infarction is a rare stroke syndrome, when compared to the dorsolateral medullary (Wallenberg) syndrome caused by posterior inferior cerebellar artery (PICA) infarction. This is because the medial medullary artery is supplied by the anterior spinal artery, which itself is formed from both vertebral arteries (allowing for protection against unilateral vertebral occlusion), whereas the PICA originates from only a single vertebral artery [10].

The most classical presentation of medial medullary stroke is Dejerine syndrome [6, 7, 11, 12, 13], which consists of the triad of ipsilateral hypoglossal palsy, contralateral hemiparesis and contralateral lemniscal (spinothalamic) sensory impairment (including reports of burning and pricking sensations in the face and limbs) [14]. However, a bilateral pattern of sensory impairment can occur, as a staggering onset [10], when there is occlusion of the common stem of bilateral medial medullary arteries. The most common eye movement abnormality is up beat nystagmus (UBN), although horizontal or more complex forms of nystagmus (e.g. seesaw or bowtie nystagmus) have been described. Other signs include truncal lateropulsion [9] (i.e. falling to one side), although for lesions that cross the midline the resultant sign is truncal ataxia [15]. In this case report we present a patient who developed Rt. Medial Medullary infarction, due to thrombosis of vertebral or anterior spinal artery, secondary to Protein 'S' Deficiency along with hypothyroidism (Hashimoto's Thyroiditis), which is very rare.

Case report

A 27 year old, female, housewife, was admitted for weakness of left sided limbs, difficulty in speaking and numbness of left side of the body of 1day duration. History of presenting illness - Patient was apparently asymptomatic a day earlier, then she developed dizziness, sudden in onset, developed during morning chores followed by one episode of sudden projectile vomiting, which contained food particles. Later she developed weakness of left sided limbs and numbness in the left side of the body which was sudden in onset. Later she developed difficulty in speaking linguals (d, l, n. r, t) which was sudden in onset. No history of abnormality in smell sensation, no history of difficulty in mastication or loss of sensation over face, no history of deviation of angle of mouth and drooling of saliva, no history of loss of taste sensation, no history of tinnitus or loss of hearing, no history of hoarseness of voice, but patient had difficulty in pronouncing linguals, no history of dysphagia, nasal regurgitation, no history of drooping of shoulders or difficulty in shrugging of shoulders, no history of weakness of rotation of head, no history of bladder or bowel disturbances and abnormal sweating, no history of head injury, loss of consciousness, loss of sensations, no history of seizure like activity, no history of bladder/ bowel incontinence, no history of bleeding or clotting disorder. No history of similar complaints in the past, history of 4 abortions in the previous pregnancies during 2nd trimester, diagnosed hypothyroid in 2019 and was on T. thyronorm 50 mcg but not taking treatment since 1 month, N/K/C/O HTN, DM,

Epilepsy, CAD, TB. There were no history of similar complaints in the family, Personal History was unremarkable.

General physical examination was unremarkable, Temp – Normal, Resp. Rate – 18/mt, PR 78/min regular in rhythm, Blood Pressure of 130/80 mmHg.

On CNS examination - Intellectual functions – normal, Dysarthria present, for linguals. Cranial nerves Olfactory nerve -able to perceive odor from both the nostrils, Optic nerve -Visual acuity: Normal, 6/6 in both eyes, Colour vision: Normal, Peripheral visual fields; by Confrontation test: Normal in all the directions, Pupils: Equal in size, and reacting to light, Pupillary reflex; Direct and indirect light reflexes present in both the eyes, Fundus exam - Both Eyes – Normal, Occulomotor, Trochlear, Abducens nerves - Conjugate movements - Horizontal conjugate movements, normal in both directions (Rt and Lt),Vertical conjugate movements, normal in both directions (Up and Down), Individual muscles - 3rd CN – sup. rectii, inf. rectii, med. rectii and inf. obliques are normal on both sides, 4th CN – sup. oblique normal on both sides, 6th CN – both. lat. rectii – normal. Trigeminal nerve, Sensory-able to perceive sensations on all   over the face, Motor-able to clench teeth, Corneal and conjunctival reflexes-intact. Facial nerve - Wrinkles on fore head are normal on both sides, no ptosis, no deviation of any angle of mouth. Vestibulocochlear nerve – Rinnes - positive, Webers - normal, Schwabachs - normal. Glossopharyngeal and Vagus nerves - uvula is in the centre, palatal and uvula movements are equal on both sides. Spinal accessory nerve - able to shrug the shoulders, equally on both sides. Hypoglossal nerve - Deviation of tongue to the right side, with fasciculations on right half, with wasting of tongue muscles on right side (**Figure – 1**).

Motor system examination - Power – lt. side – upper limb - 2/5, lower limbs – 3/5, Tone – hypotonia in lt. sided limbs, Superficial reflexes - plantars - left up going, others – normal, Deep reflexes – exaggerated on lt. side, Sensory system – Superficial sensations – touch, pain- present all over, Deep sensations – vibration and position – absent on lt. side, Cortical sensations – able to perceive Tactile localization, Stereognosis, Graphasthesia, Cerebellar system - No hypotonia, No Ataxia, no Swaying, No scanning speech, Finger Nose Finger Test could not be done on Lt. side (Power 2/5), Rt. side – normal, Heel Knee Test: normal on both sides, Able to perform alternate rapid movements. Vertical upbeat Nystagmus was present in both the eyes. No vasomotor changes, No trophic ulcers, Exam of other systems – normal, and there was no thyromegaly.

Figure - 1: Wasting of right tongue muscles.

On Investigation - Hb-12.1g%, RBC count 7.2 mill/cumm, WBC count 6500 cells/cumm with Neutrophils - 80%, Lymphocytes - 24%, Eosinophils - 2%, Basophils - 0%, Monocytes - 2%, Platelet count – 1.9 lakhs/cu mm. ESR 1st hr - 20 mm, Blood Urea 20 mg%, Serum Creatinine - 0.6 mg, Complete Urine Examination (CUE) – normal, Random Blood Sugar - 103 mg/dl, Serum Electrolytes: Na – 143 m mol/L, K - 3.6 m Eq/L, CL – 109 mEq/L.

LFT – Normal, Fasting Lipid Profile-WNL, ESR-10 mm/hr, Carotid Doppler-Normal, ECG-Sinus Tachycardia, 2dECHO-Normal,THYROID PROFILE - T_3-1.11 ng/ml, T_4-8.37 microgm/dl, TSH-12.17 microIU/ml, Anti TPO antibodies-68.07 IU/ml(elevated), Anti Tg Ab – 60 IU/ml. FNAC of thyroid revealed autoimmune thyroiditis. USG of Thyroid – normal, USG abdomen revealed normal study. APTT-23.8, PT-16.0, INR-1.2, Anti dsDNA - 21.76IU/mL (<100=Negative,>/=100= Positive), ANA Screening-Negative, Phosphilipid antibody IgM and IgG-Negative, Anti Thrombin III activity-92%(N= 80 – 120%), Protein C activity-84%(N= 70 – 130%), Protein S activity-40% (N= 55 – 123%), S. Homocysteine = < 15 micro moles/lt, Complement-C_3-1.02g/L (N=0.90-1.8g/L), Complement C_4-0.317g/L (N=0.1-0.4g/L), MRI was done (**Figure – 2, 3**).

Figure – 2: MRI – (A) DWI AND (B) ADC – reveals right medial medullary infarction.
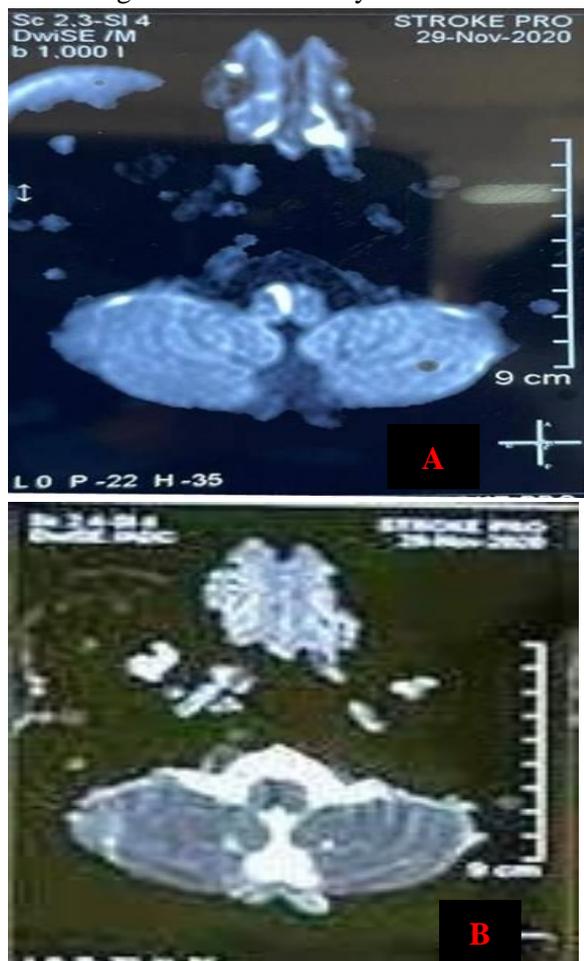
Figure – 3: MRI Angiogram – **(A)** incomplete circle of Willis, absent posterior communicating artery (arrows); **(B)** hypoplastic right P1 segment (arrow).
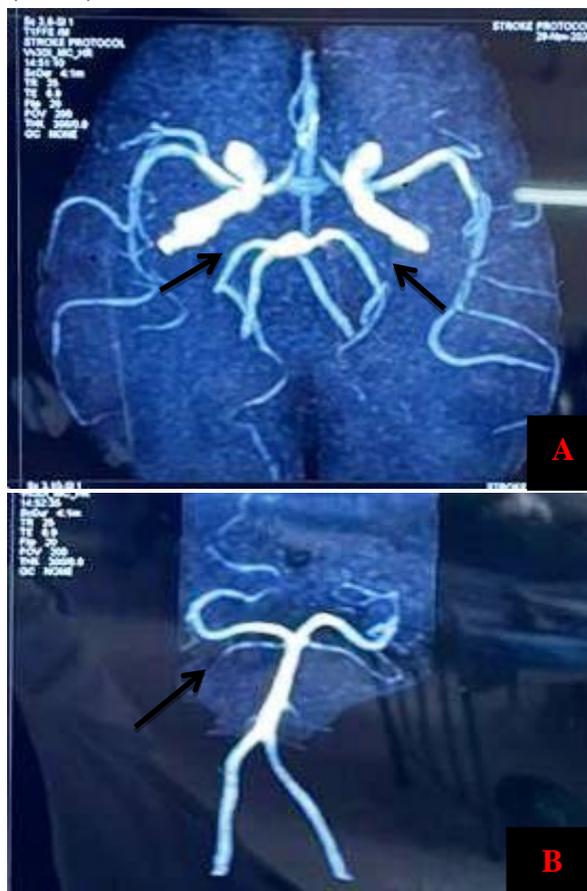
A diagnosis of Rt. Medial Medullary Syndrome with Upbeat Nystagmus due to Protein 'S' Deficiency and Hypothyroidism (Hashimatos Thyroiditis) was made.

Treatment

1) Tab. Ecosprin 75 mg 1 OD,
2) Tab. Eltroxin 75 mcg OD BBF
3) Physiotherapy

The hemiparesis completely improved, but vertical nystagmus was persisting on follow up after one month.

Discussion

Pathophysiology

After establishing the diagnosis of MMS one has to search for the cause and pathophysiology for the management of the case. The common causes of acute brainstem dysfunction are arterial

thrombosis, infection i.e. meningoencephalitis and autoimmune disorders like multiple sclerosis or paraneoplastic syndrome, neoplasm or Wernicke's encephalopathy [15].

Different causes of medullary infarction have been documented including thrombosis, artery to artery embolism, talc or fibrocartilaginous embolism, syphilitic arteritis, fibromuscular dysplasia, and dissection of the arterial wall. In young individuals, dissection of the vertebral artery may develop spontaneously or after chiropractic manipulation, yoga, or trauma to the neck. The presence of severe headache or neck pain should raise the suspicion of vertebral artery dissection [12].

Assessment of Lesion Pattern and Etiologies Distribution of Infarcts Based on MRI Findings

The lesions may be vague initially, as the time elapses the lesions become apparent. The lesions were categorized, rostro-caudally as "rostral," "middle," and "caudal," the majority of lesions were located in the rostral medulla , lesions were ventro-dorsally classified according to the diagram of the upper medulla (**Figure - 4**) as: "ventral (V)" (ventral part, presumably containing the pyramid); "middle (M)" (middle part, presumably including the medial lemniscus); and "dorsal (D)" (dorsal part, presumably including the medial longitudinal fasciculus [MLF] in a lesion extending to the dorsal surface of the medulla) (Figures 4) [9].

Figure – 4: Schematic diagram showing structures in the rostral medulla, and the ventral (V), middle (M), and dorsal (D) portions [9]
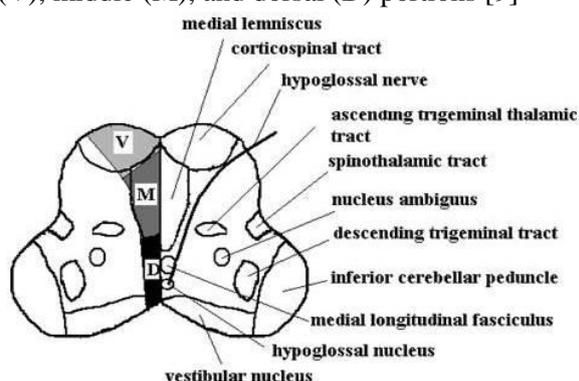
Evaluation of Arterial Stenoses [9]

The degree of arterial stenosis was categorized into mild (less than 50% diameter reduction), moderate (50% diameter reduction with complete distal flow), severe (segmental non-visualization of artery), occlusion, and aplasia (non-visualization of the entire VA)/hypoplasia (diffuse homogeneous narrowing of the entire VA). The non-visualization or homogeneous narrowing of the distal VA after the origin of the posterior inferior cerebellar artery interpreted as aplasia/hypoplasia, and irregular narrowing interpreted as atherosclerotic vascular stenosis [9].

Presumed Stroke Mechanisms [9]

The presumed stroke mechanisms were categorized with modification of recent guidelines.

1. Large vessel disease (LVD), when there was a significant stenosis or occlusion of the relevant artery (VA) that explains the infarction. The LVD was divided into 3 sub categories:
(A) atheromatous branch occlusion (ABO)
 when infarcts were on the territory of one or a few perforating branches arising from stenosed (of any degree) or occluded distal, intracranial VA, or VA-basilar artery (BA) junction that presumably occluded the orifice of perforators;
(B) artery-to-artery embolism (AAE)
when there was a moderate to severe stenosis or occlusion in the proximal VA with no distal VA disease. Because the degree of stenosis may be exaggerated in V1 segment, only severe stenosis or occlusion was considered as "significant" in V1. Also, if there was any possibility of pulsation or motion artifacts in the V1, we did not consider the lesion as significant;
(C) AAE+ABO when there are stenosis or occlusion in both distal and proximal VA.
2. Cardiogenic embolism (CE):
CE was determined when there was emboligenic heart disease, without significant atherosclerosis.
3. Small vessel disease (SVD):
SVD was defined when patients had (1) hypertension or diabetes; (2) no emboligenic heart disease; (3) normal angiogram findings.

4. Undetermined etiology

was defined when there was (1) presence of 2 or more causes; (2) imaging findings that were hard to differentiate between vascular lesion and hypoplasia/aplasia of VA [9].

Other medullary syndromes [12]

Apart from lateral medullary syndrome and medial medullary syndromes, there are other ischemic vascular syndromes of the medulla, namely bilateral medial medullary, hemimedullary, and bilateral lateral medullary syndromes (**Figure - 5**) [12].

Figure – 5: Combined medullary syndromes (shaded areas indicate location of lesions) [12]
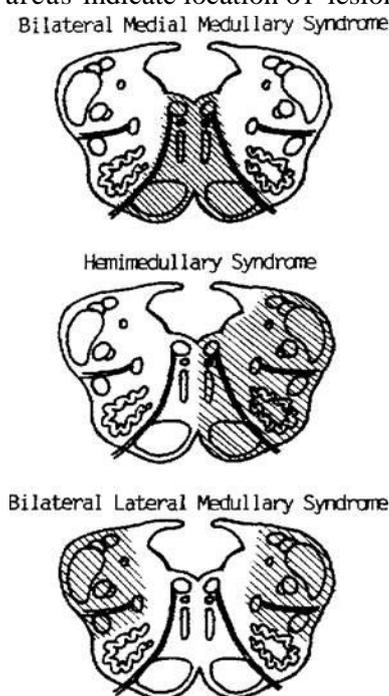
These syndromes are better viewed as combinations of the major syndromes occurring either simultaneously or during separate episodes. As medial medullary syndromes are rare, these combination syndromes are even more rare. Bilateral medial medullary syndrome was among the cases described by Dejerine in his book in 1914 [3]. Davison provided the first clinicopathological description of this syndrome in 1944 [16]. In the review of Ho and Meyer [17], bilateral involvement occurred in 7 of 15 cases of medial medullary syndrome. Clinically, it presents with flaccid quadriplegia sparing the face, bilateral disturbance of deep sensation, hypoglossal nerve palsy, and respiratory failure. Combinations of complete and atypical medial medullary syndromes may give slightly different findings in bilateral medial medullary infarctions (**Table - 1**). In hemimedullary infarction, the clinical diagnosis depends on recognizing features of both the medial and lateral medullary infarctions. Reinhold, reported the earliest clinicopathologically proven case of hemimedullary syndrome in a physician in 1894, a year before Wallenberg's description of the lateral medullary infarction. Babinski and Nageotte in 1902 described a medullary syndrome that bears their names and presents with lateral medullary syndrome and contralateral hemiparesis or an extensor plantar response. This is essentially a combination of lateral medullary infarction and "incomplete" medial medullary syndrome presenting as pure motor hemiparesis. In one recent review of hemimedullary syndromes, only six autopsy-proven cases were collected and one more added by MRI correlation. The term "hemimedullary syndrome" should probably be reserved for cases in which the lateral and medial infarctions occur simultaneously [1].

Medial medullary infarction (MMI) is characterized by a triad of contralateral hemiparesis, decreased position and vibration sensation in the contralateral side of the body, and ipsilateral tongue paralysis [18]. MMI generates distinct patterns of ocular motor abnormalities in contrast to those observed in lateral medullary infarction (LMI). The horizontal nystagmus beats toward the lesion. Gaze evoked nystagmus (GEN) is more intense on looking ipsilaterally [19]. The mechanisms of spontaneous nystagmus and GEN in MMI can be explained by damage to the nucleus prepositus hypoglossi (NPH) [19, 20, 21]. Upbeat nystagmus in MMI has been ascribed to lesions involving the perihypoglossal nuclei [19]. However, NPH lesions do not generate upbeat nystagmus in monkeys [21]. Instead, evolution of upbeat into hemiseesaw nystagmus with resolution of a unilateral lesion, as previously

observed in a patient with bilateral MMI, suggests an involvement of the vestibulo-ocular reflex (VOR) pathways from both anterior semicircular canals (SCCs) as a mechanism of upbeat nystagmus [22].

Table – 1: Presentations in bilateral medial medullary syndrome [12].

Medial medullary syndrome	Findings
1. Bilateral complete syndrome	Quadriplegia, total paralysis of tongue, bilateral impairment of deep sensation
2. Unilateral complete syndrome + contralateral syndrome without tongue paralysis	Quadriplegia, tongue deviation to one side, bilateral impairment of deep sensation
3. Unilateral complete syndrome + contralateral syndrome of pure motor hemiparesis	Quadriplegia, tongue deviation to one side, with contralateral impaired deep sensation
4. Bilateral syndrome without tongue paralysis	Quadriplegia, tongue midline on protrusion, bilateral impairment of deep sensation
5. Unilateral syndrome without tongue paralysis + contralateral syndrome of pure motor hemiparesis	Quadriplegia, tongue midline on protrusion, unilateral impairment of deep sensation
6. Bilateral syndrome of pure motor hemiparesis	Quadriplegia, tongue midline on protrusion, no deep sensation impairment

Because the MLF is a midline structure that carries signals from the vestibular to the ocular motor nuclei, upbeat nystagmus in unilateral lesions may be explained by concurrent damage to decussating fibers from both anterior SCCs at the rostral medulla. The cell groups of the paramedian tract (PMT), which are involved in processing of vertical eye position through their projections to the cerebellar flocculus, may be another neural substrate for upbeat nystagmus in MM [23]. Ocular contrapulsion also has been described in MMI and is explained by damage to the climbing fiber before decussation around the hilus of upper part of the inferior olivary nucleus (ION) in the rostral medulla [18]. Patients with MMI may also show contraversive ocular tilt reaction (OTR), and subjective visual vertical (SVV) tilt [19, 22, 24].

Vertical nystagmus

Patients with UBN can have lesions to the midbrain, affecting the interstitial nucleus of Cajal [25], the pontine ventral tegmental tract [26],or (as is the case here) paramedian dorsal lesions in the medulla, affecting the nucleus intercalatus of Staderini [15, 27]. The pathophysiology of spontaneous upbeat (UBN) and downbeat (DBN) nystagmus is reviewed in the light of several instructive clinical findings and experimental data. UBN due to pontine lesions could result from damage to the ventral tegmental tract (VTT), originating in the superior vestibular nucleus (SVN), coursing through the ventral pons and transmitting excitatory upward vestibular signals to the third nerve nucleus. A VTT lesion probably leads to relative hypoactivity of the drive to the motoneurons of the elevator muscles with, consequently, an imbalance between the downward and upward systems, resulting in a downward slow phase. The results observed in internuclear ophthalmoplegia suggest that the medial longitudinal fasciculus (MLF) is involved in the transmission of both upward and downward vestibular signals. Since no clinical cases of DBN due to focal brainstem damage have been reported, it may be assumed that the transmission of downward vestibular signals depends only upon the MLF, whereas that of upward vestibular signals involves both the MLF and the VTT. The main focal lesions resulting in DBN affect the cerebellar flocculus and/or paraflocculus. Apparently, this structure tonically inhibits the SVN and its excitatory efferent tract (i.e. the VTT) but not the downward vestibular system. Therefore, a floccular lesion could result in a disinhibition of the SVN–VTT pathway with, consequently, relative hyperactivity of the drive to the motoneurons of the elevator muscles, resulting in an upward slow phase. UBN also results from lesions affecting the caudal medulla. An area in this region could form part of a feedback loop involved in upward gazeholding, originating in a collateral branch of the VTT and

comprising the caudal medulla, the flocculus and the SVN, successively. Therefore, it is suggested that the main types of spontaneous vertical nystagmus due to focal central lesions result from a primary dysfunction of the SVN–VTT pathway, which becomes hypoactive after pontine or caudal medullary lesions, thereby eliciting UBN, and hyperactive after floccular lesions, thereby eliciting DBN. Lastly, since gravity influences UBN and DBN and may facilitate the downward vestibular system and restrain the upward vestibular system, it is hypothesized that the excitatory SVN–VTT pathway, along with its specific floccular inhibition, has developed to counteract the gravity pull. This anatomical hyperdevelopment is apparently associated with a physiological upward velocity bias, since the gain of all upward slow eye movements is greater than that of downward slow eye movements in normal human subjects and in monkeys.

This circuit could specifically be involved in the upward vestibular system, and does not appear to have an equivalent in the downward system; the result could be a slight upward velocity bias in the normal state [28]. Infarction of the medial medulla, leading to medial medullary syndrome (Dejerine's), is a rare condition that comprises < 1% of all ischemic strokes in posterior circulation [7]. It is classically defined by the presence of Dejerine's triad of contralateral weakness in upper and lower extremities (with facial sparing) due to pyramidal tract involvement, contralateral hemisensory loss of vibration and proprioception due to medial lemniscus involvement, and ipsilateral tongue weakness, due to CN12 nucleus involvement [29].

This syndrome results from occlusion of the vertebral artery or one of its medial branches. The largest clinical study of MMI to date examined 86 patients with MRI-proven MMI and found considerable variability in symptoms, with the most common being hemiplegia (91%), vertigo/dizziness (59%), impaired vibratory sense (56%), impaired proprioception (48%),

nystagmus (44%), dysphagia (29%), nausea/vomiting (16%), and headache (10%) [9].

Despite the eponymously named Dejerine triad of symptoms, ipsilateral tongue weakness is a rare finding (<3%) [9, 30]. This is likely because tongue weakness is due to lateral medullary involvement and is not seen in the overwhelming majority of patients with small, paramedian infarcts [5].

Neuroimaging - Role of MRI

The clinical impression of brainstem dysfunction was not corroborated on (initial) brain MRI, a fact that may have thrown the unwary clinician off the scent. However, a normal brain MRI does not exclude organic brain disease and, in particular, limitations of the sensitivity of brainstem imaging with MRI must be appreciated. Several case series looking at the diagnostic accuracy of MRI in detecting brain infarctions suggest that up to one-third of brainstem infarcts (especially in the medulla) can be missed on initial MRI, but can be detected on repeat MRI [38, 39]. This compares with an MRI false-negative rate of approximately 5% across all brain infarct locations [40]. The reasons behind false-negative diffusion weighted MRI scans pertain to resolution capacity of diffusion MRI [40], susceptibility artefacts that cause distortions in brain stem imaging [41], and the fact that reduction in blood flow may be above the threshold for diffusion-related effects and yet severe enough to cause neuronal dysfunction [15, 42, 43]. Although DWI is sensitive to acute stroke approximately 92% of the time, the detection rate of posterior circulation strokes may only be half as high as that in anterior circulation [31, 32]. One possible explanation for this is that the brainstem is a relative "blind spot" for radiologists [33]. Compared to cerebral infarcts, brainstem lesions are often harder to detect because they are smaller, may lack pronounced mass effect, and have more densely compact anatomy, with close proximity of vascular, bony, neural, and soft tissue structures [33]. Moreover, medulla oblongata

infarctions,are the least detectable, even compared with other brainstem regions [34].

Although DWI is the most sensitive method of detecting acute ischemic stroke, cross-sectional CT angiography and CT perfusion (CTP) have emerged as useful adjuncts in evaluating posterior circulation infarcts [35, 36]. CTP can further improve infarct detection and identify salvageable brain tissue (penumbra) more rapidly than MRI alone. As a result, it can potentially prolong the window for intravascular therapy and improve functional outcomes [35, 36]. CTP is an especially useful adjunct for evaluating posterior fossa infarcts, which are notoriously difficult to detect [36, 37].

Prognosis

In the medial medullary syndrome, the outcome is favorable when the lesion is limited to the upper third of the medulla. A higher incidence of respiratory compromise has been reported in medial infarcts occurring in the lower two-thirds of the medullary pyramid. Furthermore, bilateral involvement is more common in the lower medulla and has a poorer prognosis [44].

The combined syndromes generally have a more complicated clinical course and carry a graver prognosis [1, 45].

Conclusion

Recognition of the patterns and characteristics of abnormal eye movements observed in brainstem lesions is important in understanding the roles of each neural structure and circuit in ocular motor control as well as in localizing the offending lesion. Numerous structures located in the brainstem generate distinct patterns of abnormal eye movements when damaged. Recognition of ocular motor abnormalities from damage of each structure allows topographic diagnosis of the lesions from various disorders involving the brainstem. Although diverse patterns of eye movements may be observed in lesions anywhere along the brainstem medullary lesions mostly present various patterns of nystagmus and

impaired vestibular eye movements without obvious ophthalmoplegia. Pontine ophthalmoplegia is characterized by abnormal eye movements in the horizontal plane, while midbrain lesions typically show vertical ophthalmoplegia in addition to pupillary and eyelid abnormalities [24]. MMIs are very rare and comprise < 1% of strokes in children [7].

Their diagnosis can be especially difficult, as the brainstem is a relative "blind spot" for many radiologists [33]. Lee K. Rousslang, et al., in their case, the diagnosis was also complicated by a significant skull base artifact on the initial MRI. Understanding of classic presentations, such as Dejerine syndrome, can help localize findings on imaging [37].

In this case report we present a patient who developed Rt. Medial Medullary infarction, due to thrombosis of vertebral or anterior spinal artery, secondary to Protein 'S' Deficiency along with hypothyroidism (Hashimoto's Thyroiditis), which is very rare.

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