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Case Report

Parinaud syndrome as an unusual presentation of intracranial hypotension

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ABSTRACT

Background: Vertical gaze palsy is a rare clinical manifestation of intracranial hypotension. The typical features of intracranial hypotension include a postural headache, dural enhancement, and low cerebrospinal fluid (CSF) opening pressure.

Case Description: We describe a case of a shunt-dependent middle-aged female with aqueductal stenosis who developed recurrent presentations of upgaze palsy with postural headaches, confirmed low opening pressure, and slit ventricles on magnetic resonance imaging (MRI) due to shunt overdrainage. Her ophthalmoplegia and headaches improved following third ventriculostomy and with increasing the shunt opening pressure to prevent excess CSF drainage.

Conclusion: Intracranial hypotension should be considered part of the differential diagnosis for patients presenting with an upgaze palsy.

Keywords: Intracranial hypotension, Ophthalmoplegia, Parinaud syndrome, Slit ventricle, Upgaze palsy, Ventriculoperitoneal shunt

INTRODUCTION

Parinaud syndrome (also called dorsal midbrain syndrome and pretectal syndrome) is primarily characterized by a supranuclear vertical conjugate gaze paralysis. Other features include upper eyelid retraction (Collier's sign), dissociated pupillary response to light (pseudo-Argyll Robertson pupil), and a convergence and accommodation palsy.[21,23] The pathogenesis of vertical gaze palsy has been well described in hydrocephalus; however, only rare reports of this presentation being associated with intracranial hypotension have been published in the literature.

In the case of hydrocephalus, the third ventricle enlarges producing direct and/or indirect pressure through displacement of the suprapineal recess causing compression of decussating posterior commissure fibers responsible for bilateral conjugate vertical gaze.[23] Areas which are important in the upgaze pathway include the rostral interstitial nucleus of the medial longitudinal fasciculus (riMLF), the interstitial nucleus of Cajal (InC), and the posterior commissure.[21] The riMLF computes impulses ascending from the vestibular system through the MLF and descending fibers from the cerebral hemisphere (mainly frontal eye field) through the pretectum to finalize gaze commands[21] [Figure 1]. It is located in the reticular formation of the rostral midbrain [Figure 2]

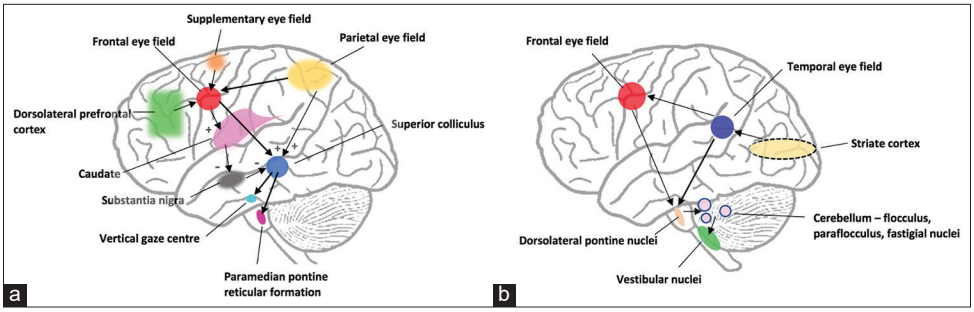

Figure 1: (a) Saccades – visual stimuli in the occipital cortex gets processed in the parietal eye field located in the posterior parietal cortex. The frontal eye field then initiates ocular motor saccades after integrating information from the parietal eye field (spatial targeting where to look), supplementary eye field and dorsolateral prefrontal cortex (latter two involved in decision making and planning movements). Direct excitation of the superior colliculus leads to the stimulation of the mesencephalic (riMLF for vertical) or pontine (horizontal) reticular formations which results in coordinated saccadic eye movement. An indirect pathway goes through the caudate and basal ganglia. The substantia nigra pars reticulata prevents saccade generation through inhibition of the superior colliculus; however, stimulation of the caudate nucleus from the frontal eye field inhibits the substantia nigra and hence this pathway. (b) Smooth pursuit – The striate cortex of the occipital lobe commences early motion analysis of visual stimuli. The temporal eye field (middle temporal and medial superior temporal areas) then integrates information to calculate velocity and direction of the object. The frontal eye field initiates motor execution after also receiving contributions from the supplementary eye field and dorsolateral prefrontal cortex which plan and track motion. Corticopontine fibers synapse on the dorsolateral pontine nuclei which project signals to the cerebellum (flocculus, paraflocculus, and fastigial nuclei), then to vestibular nuclei which modulate and coordinate cranial nerve nuclei for extraocular movement. This allows tracking of an object with constant feedback to adjust speed and direction of ocular movements so smooth pursuit of an object can occur. Note gaze centers are not part of this pathway.

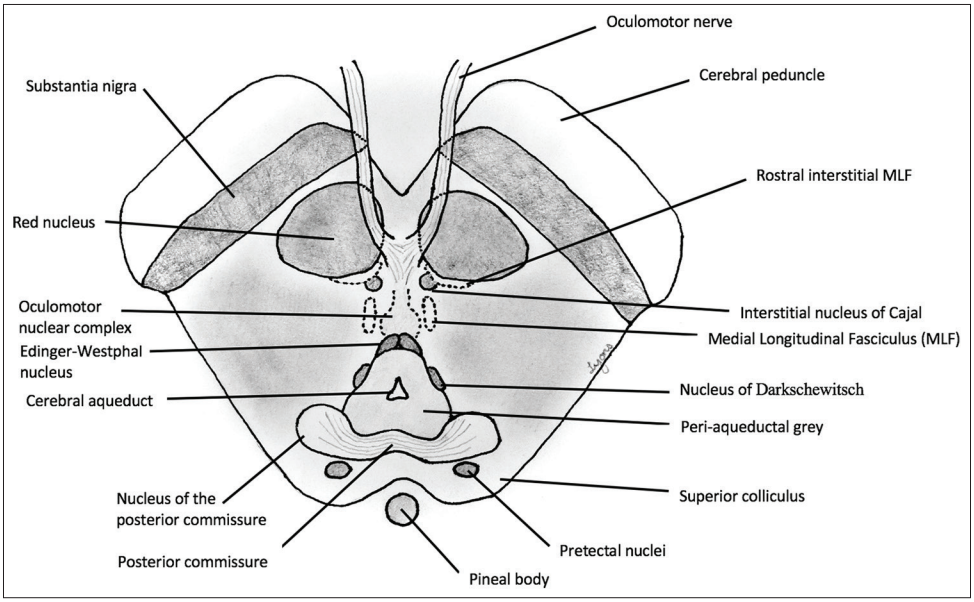

Figure 2: Cross-sectional illustration of the midbrain at the level of the superior colliculus.

and is responsible for saccadic innervation of the oculomotor and trochlear nerve with pathology causing loss of vertical saccades (up or down going).[13] The InC is a continuation of the riMLF and is responsible for stabilizing far upward gaze with lesions causing gaze evoked nystagmus.[13] The posterior commissure is located between the pineal gland and superior colliculus and coordinates vertical eye movements, predominantly upgaze.

Along with medial and superior vestibular nuclei, the perihypoglossal nuclei are thought to have an influence on the integration of vertical eye movement commands. It receives fibers from the paramedian pontine reticular formation, vestibular nuclei, and the nucleus of Darkschewitsch and has both direct and indirect excitatory influence on ocular motor neurons.[16] The nucleus of Darkschewitsch (accessory oculomotor nucleus) has also been implicated in modulation

of vertical eye movement.[1,23] It receives fibers from the medial longitudinal fasciculus and cerebellum and projects to the inferior olivary nucleus. The inferior olivary nucleus receives excitatory stimuli from the nucleus of the optic tract (one of the pretectal nuclei) and inhibitory stimuli from the perihypoglossal nuclei.[3] It also receives fibers from the spinal cord. The nucleus of the optic tract is chiefly responsible for optokinetic nystagmus and sensing retinal error during smooth pursuit and relaying this information for corrective eye movement through the inferior olive.[18] The inferior olive sends olivocerebellar fibers through the contralateral inferior cerebellar peduncle to the neocerebellum, which then projects back to pontine nuclei through the middle cerebellar peduncle in a feedback circuit to improve accuracy of voluntary ocular movements and modify eye movements in accordance with movements of the head and body in space.

The cerebellum has contributions affecting vertical eye movements. The fastigial nuclear complex receives vestibulocerebellar fibers and projects back to the vestibular nuclei through the cerebellovestibular tract. It likely has involvement in generation and execution of vertical saccades. Experimental lesions of this area have resulted in oblique trajectory of attempted vertical eye movement.[11] Interestingly with human functional imaging, the flocculus activates for downgaze however has no activation in upgaze.[11] A similar occurrence happens with lesions of the nodulus and central uvula of the cerebellum causing a marked impairment of downward pursuit, however only a minor effect on upgaze pursuit.[11]

Other supranuclear pathologies which affect structures involved in vertical gaze include progressive supranuclear palsy, multiple sclerosis, pineal tumor, vascular accidents (posterior thalamo-subthalamic paramedian artery supplies bilateral riMLF territories), parkinsonism, encephalitis, and drugs (carbamazepine and barbiturates).[21] Bilateral nuclear or peripheral nerve/muscle pathologies can also present as an upgaze palsy, but supranuclear causes can be distinguished apart as they can be overcome by the vestibulo-ocular reflex and doll's eye maneuver.[21]

Consideration of vertical gaze deficits in relation to the anatomical region of the midbrain is another approach to reviewing pathology [Figure 3]. A pathological analysis of lesion location in vertical gaze palsies was conducted in 1974 that found upgaze palsies predominantly resulted from lesions affecting bilateral pretectum, posterior commissure, or dorsal midbrain tegmentum.[23] A unilateral lesion from this study only produced an upgaze palsy when it involved the posterior commissure.[23] The tectum contains the superior colliculus which receives projections of the fronto-mesencephalic saccadic pathway and the posterior commissure which contains decussating fibers for mainly upgaze. The tectum, which is vulnerable to compressive lesions such as pineal tumors, is, hence, regarded as primarily causing upward gaze deficits. Tegmental pathology is said to be more susceptible to damage the pathways associated with the initiation of downward gaze. For example, progressive supranuclear palsy initially involves the tegmental region ventral to the periaqueductal grey, with early involvement of the riMLF, and is classically associated with inability of downward gaze. The riMLF pathway runs in the tegmentum and has bilateral innervation for upgaze to ocular elevator muscles (superior rectus and inferior oblique – oculomotor nerve), whereas innervation for ocular depressors in downgaze (superior oblique, inferior rectus – oculomotor and trochlear nerve) is unilateral.[8] Therefore, a unilateral lesion will affect downgaze. Bilateral riMLF lesions may also affect downgaze more than upgaze, given each riMLF supplies both oculomotor nuclei for the upgaze pathway, upgaze has a greater chance of remaining intact.[8] Downgaze fibers are thought to run in the lateral riMLF, whereas upgaze fibers run in the medial riMLF, and hence, midline compressive lesions are more likely to affect the upgaze pathway.[12]

Despite it being generally useful to use the paradigm, whereby downgaze represents tegmental pathology and upgaze represents tectal pathology,[12] pitfalls occur given the extensive integration of pathways for vertical gaze. The InC fibers for upgaze and downgaze cross in the posterior commissure, and hence, compression of the posterior commissure (located in the tectum) can produce both up and downgaze deficits. A study of ophthalmoplegia in 30 patients with progressive supranuclear palsy found an upgaze (rather than expected downgaze) limitation (47%), next common was bilateral up and downgaze (30%) while only 23% had a downgaze palsy.[14] Reverse Parinaud syndrome is a rare condition that has also been described where a convergence palsy and light-near dissociation are evident, however, patients present with a downgaze palsy

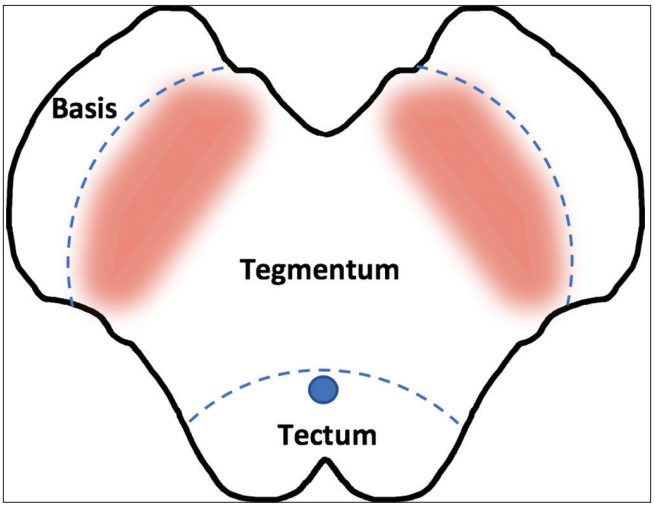

Figure 3: Cross-sectional image demonstrating regions of the midbrain.

instead of upgaze.[15] The variations in clinical presentation may represent anatomical variation or despite significant advances in vertical gaze pathway mapping, perhaps, not all pathways are fully appreciated. Therefore, while the tectum upgaze and tegmental downgaze principle is a useful clinical guide to the localization of pathology, it is wise to remember that exceptions to the rule occur and imaging is the gold standard.

This report describes a case of a middle-aged female with recurrent presentations of partially reversible Parinaud syndrome associated with intracranial hypotension due to shunt overdrainage.

CASE REPORT

A 47-year-old female presented with a 2-week progressive history of blurred vision, horizontal diplopia, inability to look upward, slowed mentation, and a postural headache on standing. There were no nausea, decreased consciousness, limb neurology, or fevers.

She was initially diagnosed with idiopathic intracranial hypertension at the age of 41 (2012); however, on later imaging, it became apparent that the underlying pathology was aqueductal stenosis. A ventriculoperitoneal (VP) shunt was inserted in 2012 and required two revisions, one in 2013 and 2017. After the 2017 shunt revision for kinked shunt tubing, she had recurrent admissions for intracranial hypotension symptoms with reproducible symptomatology of blurry vision, cognitive decline, postural headache, and an upgaze palsy. Shunt interrogation confirmed that the shunt was working in early 2018, so the shunt valve pressure was consequently increased to 7 to reexpand the ventricles to a normal size. This adjustment, however, resulted in acute ventriculomegaly with a drop in Glasgow Coma Scale indicating shunt dependence, so the setting was reduced back to 4 which improved her conscious state and symptoms, with her ventricles returning to slit-like size on CT. On MRI later in 2018, she was found to have new fluid-attenuated inversion recovery (FLAIR) signal changes in bilateral optic nerves and the midbrain [Figure 4], SWI and DWI were normal. This was associated with a transient reduction in visual acuity. There was slight improvement of the slit ventricles with an underlying aqueductal stenosis/web with no cerebrospinal fluid (CSF) flow void on high-resolution T2 sequences. She subsequently underwent endoscopic third ventriculostomy and external ventricular drain placement with an opening CSF pressure of 4 cm H_2O (confirming low intracranial pressure), which gave her temporary improvement of her symptoms.

Other medical history included anxiety, depression, and asthma. She was taking no regular medications, however, self-ceased fluoxetine a couple of months ago due to fatigue side effects. There were no allergies. She is nonsmoker with occasional alcohol intake. Occupation is a phlebotomist.

On examination, she was found to have an almost complete upgaze paralysis bilaterally with torsional nystagmus (more prominent in the left eye) and painful convergence-retraction nystagmus on attempted upgaze. The upgaze palsy could be overcome with the doll's eye maneuver. At rest, there was bilateral inferior deviation of her eyes. There was a convergence and accommodation paresis. The patient experienced horizontal diplopia maximally when trying to accommodate and look up. Her left eye had exotropia on upgaze; however there was no strabismus at rest. Smooth pursuit extraocular movements horizontally and downwards were normal. Pupils were equal and reactive to light, there was no light-near dissociation or relative afferent pupillary defect. Ophthalmologist review revealed no papilledema or refractive error causing the blurred vision. Intraocular pressures were normal, visual acuity 6/6 bilaterally. The rest of the cranial nerve examination was normal, as was limb neurology. There was no postural drop in blood pressure.

CSF culture had no growth, protein was low at <0.07, otherwise, cell counts and glucose were normal. Blood tests were normal including a pituitary panel with the exception of gonadotropins showing a perimenopausal pattern. Shunt series XR showed no disconnection of the shunt or CSF collection in the abdomen. CT brain showed slit lateral and third ventricles, no intracranial hemorrhage and confirmed that the tip of the catheter was in the ventricle. MRI brain showed no ischaemia, tumour, or lesions in the area of the dorsal midbrain. There were no FLAIR changes or evidence of mechanical distortion from brain 'sag' [Figures 5 and 6].

Clinically, she has Parinaud syndrome. The convergence insufficiency and accommodation palsy were likely responsible for her blurred vision and diplopia. The differential diagnosis

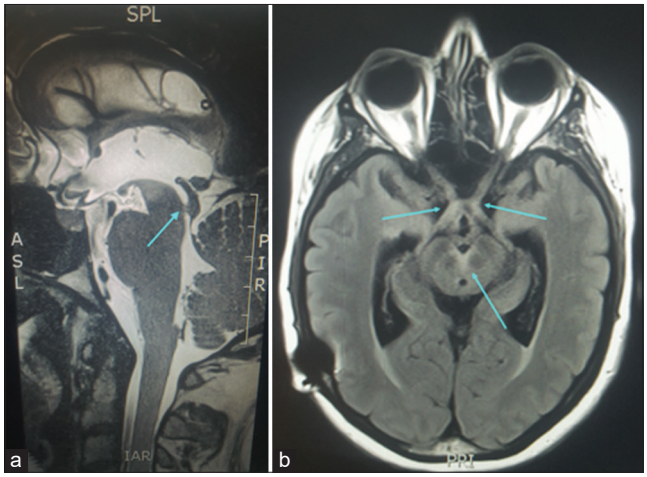

Figure 4: Previous MRI (a) High-resolution T2 MRI showing cerebral aqueduct stenosis/web. (b) Axial FLAIR MRI showing abnormal signal in the optic chiasm extending to bilateral optic tracts and in the midbrain from the interpeduncular fossa extending between the red nuclei to the area of the oculomotor nuclei.

was between an overdraining shunt and an underdraining/malfunctioning shunt in the context of slit ventricle syndrome. The final diagnosis of overdrainage causing intracranial hypotension was based on the postural symptoms which correlated with low CSF opening pressures, a functioning shunt, lack of papilledema, and the MRI finding of slit ventricles. During her admission, the VP Codman Certas shunt settings were changed from 4 to 5 which gave her marked improvement with nystagmus, postural symptoms, and the ability to move her eyes above midline when looking up, however, not full resolution of the upgaze palsy [Figures 7-9].

DISCUSSION

The characteristic diagnostic triad of intracranial hypotension is a positional headache (exacerbated by upright posture and coughing), dural enhancement with gadolinium, and low CSF opening pressures.[9] CSF has a low specific gravity (1.007) which, through Archimedes principle, creates a buoyancy force acting on the brain and hence reduces the effective weight of the brain from 1500 g to 50 g as it is suspended in CSF.[10] Reduced CSF volume (worse when standing as CSF will pool with gravity into the spinal canal) can cause the brain to sag. In particular, downward displacement of the brain is seen in the brainstem with pons flattening and the splenium with distortion of the tectal plate and decent of the midbrain.[9,19] With increased stretch from brain "sag," tearing of bridging veins can occur leading to spontaneous subdural hematomas.

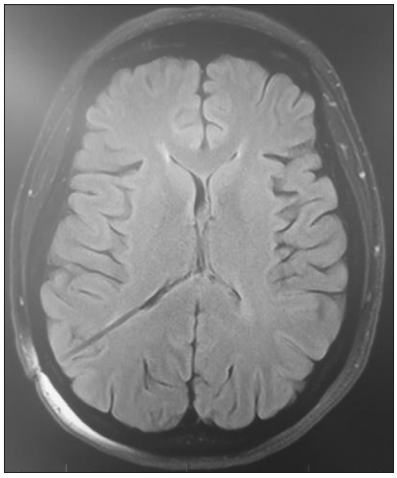

Figure 5: Preshunt adjustment axial T1 MRI demonstrating slit ventricles suggestive of shunt overdrainage.

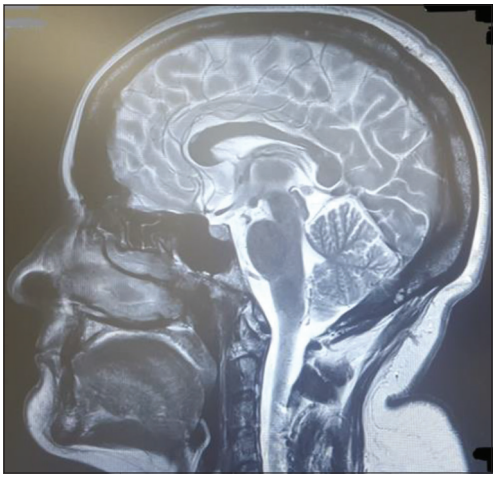

Figure 6: Preshunt adjustment sagittal T2 MRI showing normal 4th ventricle and cistern spaces, no distortion of brainstem or splenium and no tonsillar herniation.

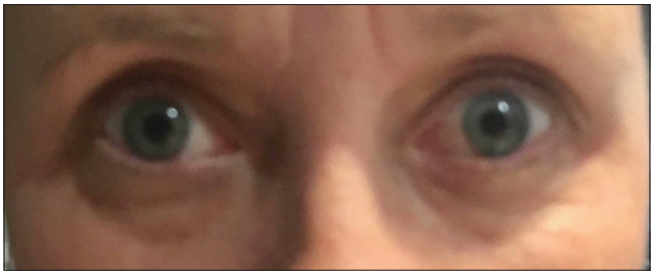

Figure 7: Five months postshunt adjustment looking straight ahead with no downward gaze preference, strabismus or ptosis. Subtle horizontal divergence.

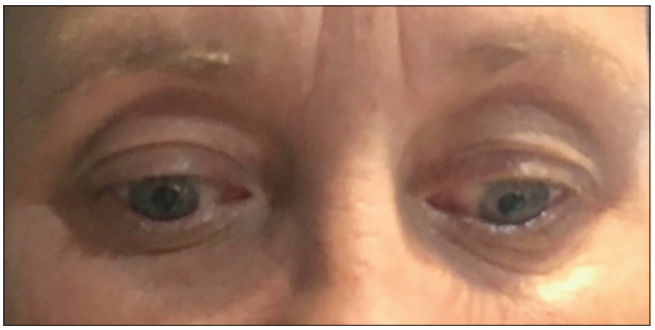

Figure 8: Five months postshunt adjustment looking down. Left eye deviation laterally.

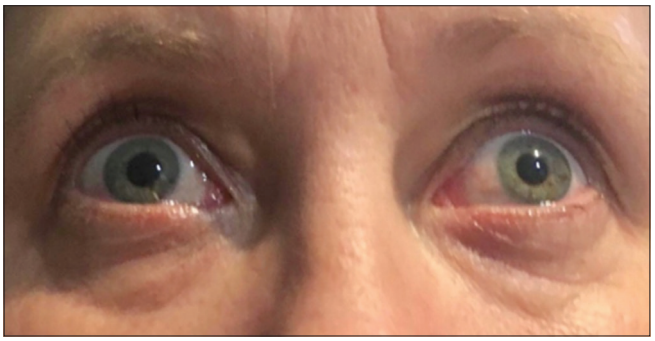

Figure 9: Five months postshunt adjustment attempting to look upward. Persistent upgaze limitation with lateral deviation of the left eye.

Subdural hygromas can also occur. Other imaging findings suggestive of this diagnosis include pachymeningeal (dural) enhancement which occurs due to increased vascular blood flow resulting in vasocongestion and interstitial edema of the dura mater in an autoregulatory response to low CSF volume to maintain intracranial pressure.[2] Enlargement of the pituitary gland may be seen and is also explained by the Monro–Kellie hypothesis, whereby CSF depletion is compensated by an increase in brain volume or volume of blood.[17]

There are three other documented cases where intracranial hypotension has presented with an upgaze palsy. Fedi *et al.* (2008) depicted a middle-aged woman with reversible Parinaud syndrome from intracranial hypotension secondary to a ruptured T11/12 perineural cyst. Gupta *et al.* (2014) presented a case of shunt overdrainage causing parkinsonism and Parinaud syndrome with persistence of the upgaze palsy despite improvement of her other ophthalmoplegia signs and postural symptoms with shunt ligation. Bray *et al.* (2016) described a case of spontaneous intracranial hypotension with associated dorsal midbrain venous infarction presenting with an upgaze palsy.[5]

The amount of brain "sag" explains some of the varying presentations of intracranial hypotension from infrequent postural headaches to stupor.[19] Vertical gaze palsy in the setting of intracranial hypotension has been rationalized by stretch or compression of the riMLF, InC, or posterior commissure due to downward displacement of the brain. Given the pattern of ophthalmoplegia in this case, the site of pathology is presumed to be either the riMLF (given the convergence palsy) or the decussating posterior commissure fibers (responsible for the predominant upgaze palsy). In this case, the MRI showed no evidence of brain sag. Perhaps, the loss of CSF volume in the third ventricle is causing an indrawing distortion and hence dysfunction of the posterior commissure fibers. The vertical gaze centers of the brain have been hypothesized to be quite sensitive to mechanical distortion,[7] and perhaps, patients with chronic CSF volume disturbances (hydrocephalus or depletion) may be more susceptible to smaller amounts of distortion either from already stretched pathways that can no longer biologically compensate or from decreased brain compliance.

The transient FLAIR changes seen in the MRI following the episode of acute hydrocephalus may have contributed to her presentation of Parinaud syndrome and decreased visual acuity on that occasion. However, they fail to explain previous and ongoing presentations of Parinaud syndrome and blurred vision without the FLAIR abnormality. FLAIR sequence suppresses CSF and intensifies T2-weighted images so abnormalities in the subarachnoid space and brain-CSF boundary become more apparent. In the context of intracranial hypotension, especially following intracranial hypertension, an elevated blood pool-to-CSF ratio can cause sulcal FLAIR hyperintensity.[22,24] The rapid decrease in CSF pressure is thought to change CSF dynamics producing the FLAIR signal.[6] There are no specific reports of optic nerve or midbrain FLAIR changes associated with intracranial hypotension; however, we have postulated that it is also related to the rapid decrease in CSF pressure/volume.

Optokinetic nystagmus is a reflex oscillation in response to motion in the visual field, whereby the body tries to stabilize an image on the retina. Optokinetic nystagmus response occurs when an object being tracked visually by the eyes reaches the limit of vision and, not being able to comfortably pursue the object anymore, the eyes rapidly return to a neutral position with a reflex saccade. There are two main pathways responsible for optokinetic nystagmus: direct retinal fibers to the pretectum (nucleus of the optic tract) and a cortical pathway mediated through the occipital lobe. A key feature of Parinaud syndrome is convergence retraction nystagmus which, with optokinetic nystagmus testing, presents as an abnormal optokinetic response which facilitates the diagnosis.[15] Convergence retraction nystagmus is an episodic refractory twitching which occurs on attempted upgaze and can often be subtle and difficult to appreciate. Examination is best elicited using an optokinetic nystagmus drum rotating downward which accentuates the convergence retraction nystagmus underpinning the diagnosis.[15] Another way to measure subtle optokinetic abnormalities is electrophysiological testing. Electrophysiologically, the quick phase of optokinetic nystagmus is reduced in Parinaud syndrome. Electrooculography involves electrodes inserted into the inner and outer canthus of the eye. It works well for horizontal eye movement abnormalities; however, recordings for vertical eye movements are greatly affected by eyelid artifact and nonlinearities, rendering it of limited value.[6] Infrared oculography involves a camera which tracks ocular movements and reliably identifies deficits (except involving torsional movements) which would markedly aid in differentiating ocular disorders and expediting diagnoses, however is not readily available.

Shunt underdrainage and overdrainage are a common result in shunt-dependent patients with fine tuning of opening valve pressures and occasionally different types of shunts required to find the right balance of CSF drainage for each individual patient. Long-term shunt overdrainage can lead to craniosynostosis in children, slit ventricle syndrome, subdural hematomas, and intracranial hypotension symptoms. There is 10–12% incidence of one of these complications occurring in the initial 6.5 years following the first shunt insertion.[20]

Slit (collapsed) ventricle syndrome is a complication of chronic shunt overdrainage. Given the depletion of CSF

within the ventricular spaces, the surface tension increases (as with collapsed alveoli in the lung) due to Laplace's law, it, hence, requires more intraventricular pressure to overcome the tension within the wall of the ventricle for expansion to occur.[4] As demonstrated by blowing up a balloon, it requires more effort/pressure initially, but is easier to blow up once the balloon is sufficiently inflated. Unfortunately, Laplace's law does not apply perfectly to the ventricular system given the original calculations are based on inert spheres. The ventricles are not spherical in configuration and the dynamic biological variation due to periventricular microanatomy means ventricular wall tension is not constant in all regions. The relationship between pressure, compliance, wall tension, and size of the ventricles is, hence, complex; however, Laplace's law is a useful equation to help understand the simplified system. Once slit ventricle syndrome is established, even with intermittent shunt obstruction, ventriculomegaly may not occur due to the inability to overcome wall tension; however, this is likely in the setting of the shunt allowing minimal (but enough) CSF drainage to prevent enough pressure build up to overcome the wall tension.[4] Due to low CSF volume, intracranial compliance decreases and consequently amplified rises in intracranial pressure can occur with shunt underdrainage. In symptomatic patients, the aim is to increase the opening pressure of the valve so that less CSF will drain and the ventricles may expand to a normal size.[4]

CONCLUSION

Intracranial hypotension should be considered part of the differential diagnosis for patients presenting with an upgaze palsy. This is especially prominent in patients with intracranial shunt devices *in situ* where overdrainage is a common phenomenon.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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