

## **Case Study**

**Title:** Blurring of Vision, Diplopia, Giddiness – Do we need to Image?

### **ABSTRACT**

**Aim:** We report a case of Parinaud syndrome secondary to an acute small left medial thalamus infarct.

**Presentation of Case:** Patient's main presenting complaint was diplopia, blurring of vision and giddiness. The main clinical manifestations were that of a severe restriction in upward gaze, and convergence-retraction nystagmus.

**Discussion:** Cases of cerebrovascular events are usually first seen by the Emergency Department where diagnoses are made and acute management is given. In this case, the patient was referred to the Eye clinic from the Emergency Department for visual symptoms. Parinaud syndrome was promptly recognised by the ophthalmologist upon examination and the decision for urgent brain imaging with referral to the neurologist was quickly made. Timely management of patient likely contributed to his favourable outcome.

**Conclusion:** This case highlights the important role of ophthalmologists in the early recognition of Parinaud syndrome, which can allow for prompt diagnosis and management. Timely diagnosis of Parinaud syndrome can be crucial to patient's outcome.

**Key words:** Parinaud syndrome, convergence nystagmus, supranuclear palsy

### **INTRODUCTION**

Parinaud syndrome is a supranuclear paralysis of the vertical gaze resulting from damage to the mesencephalic tectum. It consists of abnormalities of eye movement and pupil dysfunction, namely vertical gaze palsies, convergence-retraction nystagmus, convergence bias with horizontal saccades, light-near dissociation, corectopia, spastic-paretic accommodation and pathological lid retraction. It results from direct or compressive injury to the dorsal midbrain, and can arise from a variety of causes, such as pineal, midbrain or 3<sup>rd</sup>

31 ventricle tumours, aqueductal stenosis, multiple sclerosis, arteriovenous malformations,  
32 encephalitis, neurosyphilis, uncal herniation, and cerebrovascular disorders.

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## 34 CASE REPORT

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36 We report an interesting case of a 35-year-old Indian male who was referred by the  
37 Emergency Department to the Department Of Ophthalmology and Visual Sciences, Khoo  
38 Teck Puat Hospital, Singapore, for an acute onset of bilateral painless blurring of vision  
39 associated with binocular vertical diplopia of 3-day duration. There was no associated focal  
40 numbness or weakness, headache, nausea or vomiting. The patient was a smoker of 8.5  
41 pack years, and was diagnosed with diabetes mellitus type 2 three years ago. He had no  
42 other known ischaemic risk factors such as hypertension, ischaemic heart disease or  
43 hyperlipidaemia, and no prior ocular history.

44

45 On examination, distant Snellen visual acuity was 6/15 for both eyes, best corrected to 6/12 in  
46 the right eye and 6/9 in the left eye. There was no relative afferent pupillary defect. Intraocular  
47 pressure was normal. Anterior and posterior segments were unremarkable. However, patient  
48 had a severe restriction of upward gaze. Eye movements were otherwise full in lateral gazes  
49 and downward gaze. Convergence-retraction nystagmus was noted. **Figure 1** shows the  
50 clinical pictures of patient's eyes in different directions of gazes.

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52 On further examination, patient had normal cranial nerve function, full power in all limbs, and  
53 intact sensation. However, he had unsteady gait and was unable to perform tandem gait.

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55 Based on these findings, a provisional diagnosis of Parinaud syndrome was made. Patient  
56 was admitted on the same day and magnetic resonance imaging and angiography of the  
57 brain confirmed an acute small infarct in the left medial thalamus, extending slightly into the  
58 left cerebral peduncle. A neurologist further confirmed the diagnosis, with additional findings  
59 of hyporeflexia in the upper limbs and areflexia in the lower limbs. Blood pressure was  
60 normal. Patient was found to have borderline hyperlipidemia with suboptimal control of his

61 diabetes. Thyroid, liver and renal function tests, as well as electrolytes, were unremarkable.  
62 Full blood counts, peripheral blood film, prothrombin time, international normalized ratio, and  
63 activated partial thromboplastin time were normal. A young stroke work-up comprising of anti-  
64 nuclear antibody, anti-double-stranded-DNA antibody, lupus anticoagulant and anti-cardiolipin  
65 antibodies were negative. Homocysteine, anti-thrombin III, activity profiles of protein C and S  
66 were normal too. Ultrasound Doppler of carotid arteries was unremarkable.

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68 Patient was started on aspirin, enoxaparin, and simvastatin. To improve control of his  
69 diabetes, physicians made adjustments to his oral hypoglycaemic agents, and provided  
70 dietary and lifestyle advice. Patient was also given counselling on smoking cessation.

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72 Over the course of his 4-day inpatient stay, patient experienced symptomatic and clinical  
73 improvement, with resolving giddiness and diplopia as his restriction in upward gaze  
74 improved. He was subsequently able to ambulate well prior to discharge.

75

## 76 **DISCUSSION**

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78 Vertical gaze palsy has been reported to be caused by lesions involving bilateral rostral  
79 interstitial nucleus of medial longitudinal fasciculus (riMLF) or unilateral posterior commissure,  
80 while convergence nystagmus is usually caused by a lesion near or within the dorsal  
81 mesencephalon.<sup>1,2</sup> Later reports demonstrated that vertical gaze palsy and convergence  
82 nystagmus can result from unilateral riMLF lesion.<sup>3-6</sup> Each riMLF contain burst neurons for  
83 both upward and downward movements.<sup>7</sup> A unilateral lesion of the riMLF may occasionally  
84 cause combined upgaze and downgaze palsies, perhaps by disrupting bilateral upward gaze  
85 excitatory and inhibitory inputs and unilateral downgaze excitatory inputs.<sup>8</sup> Fibres responsible  
86 for upward gaze cross rostrally and posteriorly between the riMLF nuclei (near the pineal  
87 body) through the posterior commissure and are subject to interruption before descending  
88 and terminating in the contralateral oculomotor nuclei. On the other hand, pathways for  
89 downward gaze project directly downward from the riMLF to the oculomotor complex. This  
90 may be a reason for the dissociation of the upward and downward gaze palsies, and the

91 reason why upward gaze appears to be affected more than downward gaze.<sup>9</sup> There have  
 92 been a few anatomo-clinical cases which demonstrated that supranuclear palsies of the  
 93 downward gaze were related to paramedian lesions of the rostral mesencephalon involving  
 94 the riMLF, the nucleus of Cajal, and/or their afferent and/or efferent pathways, while  
 95 supranuclear palsy of the upward gaze was related to lesions of the posterior commissure.<sup>1</sup>

96

97 Convergence-retraction nystagmus was reported as a classic finding in extensive and  
 98 bilateral lesions of the dorsal midbrain, which are usually neoplastic and signal poor  
 99 prognosis.<sup>10</sup> However, in our case, while the patient did have convergence-retraction  
 100 nystagmus, he did not have extensive or bilateral lesions of the dorsal midbrain and had  
 101 instead, a good functional recovery.

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103 Benign course with favourable outcome has been reported with conservative management for  
 104 cerebrovascular events.<sup>10,11</sup> On the other hand, surgical treatment such as transpositions and  
 105 inferior rectus recessions have also demonstrated improvement in upgaze limitation and  
 106 convergence-retraction nystagmus.<sup>12</sup> Parinaud syndrome associated with brain tumours in  
 107 pineal gland or midbrain is seen more frequently in younger patients, while in older patients, it  
 108 is more commonly associated with cerebrovascular disorders such as haemorrhage,  
 109 ischaemia and infarct. In our case study, although the patient was young, he had a few  
 110 ischaemic risk factors such as diabetes and a smoking history. Borderline hyperlipidaemia  
 111 was only detected after investigations. He had no autoimmune or thrombophilic disorders. He  
 112 experienced symptomatic and clinical improvement with non-invasive and conservative  
 113 management.

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115 Cases of cerebrovascular events are usually first seen by the Emergency Department where  
 116 diagnoses are made and acute management is given. In this case, the patient was referred to  
 117 the Eye clinic from the Emergency Department for visual symptoms. Parinaud syndrome was  
 118 promptly recognised by the ophthalmologist upon examination and the decision for urgent  
 119 brain imaging with referral to the neurologist was quickly made. Timely management of  
 120 patient likely contributed to his favourable outcome.

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## 122 **CONCLUSION**

123

124 This is a case of Parinaud syndrome secondary to an acute small left medial thalamus infarct  
 125 which extended slightly into the left cerebral peduncle. The main presenting complaint was  
 126 diplopia with blurring of vision, and the main clinical manifestations were that of a severe  
 127 restriction in upward gaze, and convergence-retraction nystagmus. This case highlights the  
 128 important role of ophthalmologists in the early recognition of Parinaud syndrome, as it may be  
 129 the sole manifestation of cerebrovascular events. Early recognition can allow for timely  
 130 referral and prompt imaging for diagnosis and subsequent management. This can be crucial  
 131 to patients' outcome.

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166

## 167 **FIGURE**

**Figure 1. Clinical pictures of patient's eyes in different directions of gazes**



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169

## 170 **FIGURE LEGEND**

171

172 **Figure 1** shows the clinical pictures of patient's eyes in different directions of gazes. He also  
173 had convergence-retraction nystagmus on attempted upward gaze. There appeared to be

174 convergence bias with horizontal saccades as well. He did not have light-near dissociation,  
175 corectopia or Collier's sign.