

References

- Liu HM, Tsai LP, Chien YH, Wu JF, Weng WC, Peng SF, et al. A novel 3670-base pair mitochondrial DNA deletion resulting in multi-systemic manifestations in a child. *Pediatr Neonatol*. 2012;53:264–8.
- Broomfield A, Sweeney MG, Woodward CE, Fratter C, Morris AM, Leonard JV, et al. Paediatric single mitochondrial DNA deletion disorders: an overlapping spectrum of disease. *J Inherit Metab Dis*. 2015;38:445–57.
- Quijada-Fraile P, O'Callaghan M, Martín-Hernández E, Montero R, García-Cazorla Á, de Aragón AM, et al. Follow-up of folic acid supplementation for patients with cerebral folate deficiency and Kearns-Sayre syndrome. *Orphanet J Rare Dis*. 2014;9:217.
- Pineda M, Ormazabal A, López-Gallardo E, Nascimento A, Solano A, Herrero MD, et al. Cerebral folate deficiency and leukoencephalopathy caused by a mitochondrial DNA deletion. *Ann Neurol*. 2006;59:394–8.
- Khambatta S, Nguyen DL, Beckman TJ, Wittich CM. Kearns-Sayre syndrome: a case series of 35 adults and children. *Int J Gen Med*. 2014;7:325–32.
- Serrano M, García-Silva MT, Martín-Hernández E, O'Callaghan Mdel M, Quijada P, Martínez-Aragón A, et al. Kearns-Sayre syndrome: cerebral folate deficiency, MRI findings and new cerebrospinal fluid biochemical features. *Mitochondrion*. 2010;10:429–32.
- Sanaker PS, Husebye ES, Fondenes O, Bindoff LA. Clinical evolution of Kearns-Sayre syndrome with polyendocrinopathy and respiratory failure. *Acta Neurol Scand Suppl*. 2007;187:64–7.
- Lee AG, Brazis PW. In Paysse EA, Patterson MC, editors. Myopathies affecting the extraocular muscles in children; UpToDate 2015. Available from: <http://www.uptodate.com> [updated 26.05.15, accessed 18.10.16].
- DiMauro S, Hirano M. Mitochondrial DNA deletion syndromes, 2003. In: Pagon RA, Adam MP, Ardinger HH, et al., editors. *GeneReviews* [Internet]. Seattle (WA): University of Washington, Seattle; 1993–2016. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK1203/> [updated 03.05.11; accessed 10.11.16].

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Isolated sixth nerve palsy: an unusual manifestation of preeclampsia[☆]



Neuropatía aislada del sexto nervio craneal. Una manifestación inusual de preeclampsia

Dear Editor:

Preeclampsia is a pregnancy complication of unknown aetiology with an estimated frequency of 2%–8%. It is characterised by hypertension after week 20 of gestation in women with previously normal blood pressure, together with proteinuria, or in the absence of proteinuria but with presence of thrombocytopaenia, kidney failure, altered liver function, pulmonary oedema, or neurological symptoms.¹ The most frequent neurological symptoms include headache and visual alterations, with isolated oculomotor palsy being exceptional.² We present the case of a primiparous woman (37 weeks pregnant) whose first neurological symptom of preeclampsia was sixth nerve palsy; we also assess the possible action mechanisms.

Our patient was a 31-year-old woman assessed in the emergency department due to progressive onset of binocular diplopia in the horizontal plane, with no headache, fever, or other focal neurological signs. The neuro-ophthalmological evaluation revealed 20/20 Snellen visual acuity in both eyes, and the eye fundus examination showed well-defined papillae and spontaneous venous pulsation; the confrontation visual field test yielded normal results; esotropia was observed in the primary gaze position with slightly impaired adduction of the left eye. No other neurological alterations were observed. The general examination detected no fever, blood pressure values of 150/90 mm Hg, and distal oedema in both lower limbs; a blood analysis revealed thrombocytopaenia with 100 000 platelets/ μ L, increased LDH levels (418 IU/L), and proteinuria (Table 1). In view of these symptoms, and considering the diagnosis of preeclampsia and findings suggestive of isolated sixth nerve palsy, we requested an MRI study before labour was induced. The MRI study ruled out an underlying intracranial lesion, and labour was induced 24 hours after the patient's admission to hospital; a healthy baby was born with no complications. After delivery, the patient presented normal blood pressure values and diplopia progressively improved, fully resolving in 48 hours.

Isolated sixth nerve or abducens nerve palsy during pregnancy is exceptional, although isolated cases have been reported.^{3–6} The action mechanism by which preeclampsia causes neuropathy remains unknown, although the absence of other findings and previous published data in the literature suggest that it may cause vasospasm of the vasa

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Table 1 Blood test at admission.

Peripheral blood		Biochemical study			Urine analysis	
Haemoglobin	11.6 g/dL	Total protein	5 g/dL	Lactate dehydrogenase	418 IU/L	Protein++
Haematocrit	56.2%	Albumin	2.7 g/dL	GOT	9 IU/L	
MCV	85.0 fL	Urea	12 mg/dL	GPT	13 IU/L	
				Glucose	95 mg/dL	
Platelets	10 × 10 ⁴ /μL	Creatinine	0.9 mg/dL			
ESR	20 mm/h	Uric acid	6.7 mg/dL			
Leukocytes	14 380/μL	Alkaline phosphatase	110 IU/L			

ESR: erythrocyte sedimentation rate; GOT: glutamic oxaloacetic transaminase; GPT: glutamate-pyruvate transaminase; MCV: mean corpuscular volume.

nervorum, causing transient ischaemia that would lead to nerve palsy.⁶ In our patient, who had no history suggesting an underlying systemic disease, presenting preeclampsia and no fever, intracranial hypertension, or underlying intracranial lesion on MR images, together with the complete resolution of symptoms postpartum, suggest preeclampsia-related sixth nerve palsy.

References

- Lambert G, Brichant JF, Hartstein G, Bonhomme V, Dewandre PY. Preeclampsia: an update. *Acta Anaesthesiol Belg*. 2014;65:137–49.
- Thamban S, Nama V, Sharma R, Kollipara PJ. Abducens nerve palsy complicating pregnancy. *J Obstet Gynaecol*. 2006;26:811–2.
- Barry-Kinsella C, Milner M, McCarthy N, Walshe J. Sixth nerve palsy: an unusual manifestation of preeclampsia. *Obstet Gynecol*. 1994;83:849–51.
- Thurtell MJ, Sharp KL, Spies JM, Halmagyi GM. Isolated sixth cranial nerve palsy in preeclampsia. *J Neuro-Ophthalmol*. 2006;26:296–8.
- Park CM, Kim SY. Abducens nerve palsy in preeclampsia after delivery: an unusual case report. *J Obstet Gynaecol Res*. 2007;33:543–5.
- Vallejo-Vaz AJ, Stiefel P, Alfaro V, Miranda ML. Isolated abducens nerve palsy in preeclampsia and hypertension in pregnancy. *Hypertens Res*. 2013;36:834–5.

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Usefulness of brain SPECT imaging in the study of recurrent hypersomnia: Kleine-Levin syndrome[☆]

Utilidad del SPECT cerebral en el estudio de la hipersomnia recurrente: síndrome de Kleine-Levin

Dear Editor:

Kleine-Levin syndrome is an infrequent neuropsychiatric disorder that manifests with recurrent, self-limited episodes of



hypersomnia, normally accompanied by behavioural alterations (hypersexuality, irritability, and aggressiveness) and such cognitive alterations as confusion and hallucinations. It presents in the second decade of life, with a higher incidence in young men (4:1). Episodes last from 1 to 2 weeks, with complete remission of symptoms. Episodes may occur several times per year, separated by asymptomatic periods.¹

We present the case of a 17-year-old patient who, after symptoms of increased bowel activity, nausea, and low-grade fever, began to present a marked increase in somnolence, sleeping up to 20 hours per day; he was admitted to the internal medicine department. During his hospital stay, he underwent a blood test, head CT scan, brain MRI scan, chest and abdomen CT scan, EEG, polysomnography, and waking EEG; none revealed significant findings. The multiple sleep latency test revealed a mean sleep latency above 10 min (14 min) and did not show REM sleep in any sleep attempt. The CSF study showed a mildly elevated protein level. Considering a possible emotional origin, the psychiatry department assessed the patient on 3 occasions, with no

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