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Case report

Novel finding of paroxysmal tonic downgaze in Loeys-Dietz syndrome: expanding neurological phenotype

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Abstract: Loeys-Dietz syndrome (LDS) is a rare connective tissue disorder caused by pathogenic or likely pathogenic variants in genes encoding the transforming growth factor beta (TGF-®) pathway components. The disease was originally characterized by a typical symptomatologic triad: Bifid uvula or cleft palate, hypertelorism and aortic aneurysm with tortuosity. However, the clinical manifestations are very varied, including vascular findings (cerebral, thoracic, and abdominal arterial aneurysms and/or dissections), skeletal manifestations (pectus excavatum or pectus carinatum, scoliosis, joint laxity, arachnodactyly, talipes equinovarus, and cervical spine malformation and/or instability), craniofacial features (hypertelorism, strabismus, bifid uvula/cleft palate, and craniosynostosis that can involve any sutures), and cutaneous findings (velvety and translucent skin, easy bruising, and dystrophic scars). Neurological involvement is very uncommon in LDS, although it has been reported that serial cases with 1q41 deletion encompassing TGF-® 2 ligand (TGFB2) are linked to neurodevelopment abnormalities. The association between LDS or 1q41 deletion and paroxysmal tonic

downward gaze (PDT) has never been described in the literature. Here, we describe the case of a 4-month-old male with LDS type 4 caused by 1q41 deletion involving TGFB2 who has neurodevelopment abnormalities and paroxysmal ocular motor events (OPEs). Thanks to polygraphic video-EEG monitoring, such ocular paroxysms are classified as "paroxysmal tonic downward gaze" (PTD), a type of nonepileptic OPE. PTD has been described in most newborns as a benign phenomenon related to an immaturity of the extrageniculocalcarine visual pathway, so more often it concerns healthy and neurologically negative newborns, although associations with central nervous system pathology have been described. This is the first report of LDS with PDT as a novel clinical finding of the compatible syndrome.

Keywords: Loeys-Dietz syndrome; 1q41 deletion; video-EEG; paroxysmal tonic downgaze; neurodevelopment abnormalities; ocular motor events

1. Introduction

Loeys-Dietz syndrome (LDS) is a rare autosomal dominant connective tissue disorder caused by pathogenic or likely pathogenic variants in genes encoding transforming growth factor beta (TGF-®) pathway signaling factors [1–5]. Approximately 75% of the cases are attributed to de novo mutations, while the remaining 25% are inherited and occur within families [6]. The classification of the disease is based on the type of genetic mutation. Patients with the TGF-® receptor I (TGFBR1) or TGF-® receptor II (TGFBR2) mutations are classified as LDS 1 and 2, patients with the decapentaplegic homolog 3 (SMAD3) mutation are classified as LDS 3, and patients with the TGF-® 2 ligand (TGFB2) mutation are classified as LDS type 4 [7]. Moreover, mutations in two additional genes, TGF-® 3 ligand (TGFB3) and decapentaplegic homolog 2 (SMAD2), have been correlated to the LDS phenotype, identifying LDS types 5 and 6 [3].

LDS was first described in 2005 [8] as a disease characterized by the presence of vascular abnormalities associated with craniofacial, musculoskeletal, osteoarticular, and cutaneous manifestations. The phenotypic spectrum is highly variable, even in members of the same family, ranging from early onset in infants and young children, with typical facial dysmorphism and severe systemic features, to isolated aortic aneurysms incidentally discovered in adults [9]. Among the clinical manifestations of Loeys-Dietz syndrome (LDS), arterial vasculopathy is typically the most severe and constitutes the primary cause of early morbidity and mortality. Aortic aneurysms frequently develop at a young age and are associated with an increased risk of rupture at an earlier stage, even when the aortic dilation is relatively small in diameter [5]. The skeletal abnormalities most frequently described are excavated chest, deficient chest, scoliosis, ligamentous laxity, arachnodactyly, and craniofacial anomalies, including hypertelorism, cleft palate, bifid uvula, craniosynostosis, and micro-retrognathia [1,10]. Ophthalmologic manifestations have been described, including blue or dusky sclerae, decreased central corneal thickness, cornea guttata, scleral thinning, myopia, amblyopia, retinal detachment, central cataract, retinal vessel tortuosity, ectopia lentis, primary peripheral retinal nonperfusion, strabismus, and hypoplasia of extraocular muscles [1,9,11–19]. Neurologic impairment is relatively uncommon in individuals with LDS. However, certain abnormalities, such as hypotonia and developmental delay, have occasionally been reported, though these occurrences are rare [20].

In this case report, we detail the clinical presentation of an infant diagnosed with Loeys-Dietz syndrome type 4, characterized by a heterozygous 5.7 Mb deletion on chromosome 1q41, encompassing the TGFB2 gene. The infant exhibits ocular paroxysmal events (OPEs), specifically paroxysmal tonic downgaze, along with neurodevelopmental abnormalities.

2. Case report

The patient was a 4-month-old male, the second-born child of non-consanguineous parents. He was delivered at 37 weeks of gestation via cesarean section due to breech presentation, following a pregnancy conceived through Intra-Cytoplasmic Sperm Injection (ICSI). The delivery was uncomplicated, and his birth weight was 2,400 grams. On the third day of life, the infant was admitted to the Neonatal Intensive Care Unit (NICU) due to hypoglycemia, episodes of desaturation, which were attributed to interstitial bronchopneumonia, and hypotonia.

Cytogenetic analysis of the boy revealed normal karyotypes. Array comparative genomic hybridization (aCGH) analysis revealed a de novo 1q41 microdeletion, encompassing 17 genes of which 6 are Online Mendelian Inheritance in Man (OMIM) genes (Figure 1). Among the gene content of the deleted region, there is TGFB2, whose haploinsufficiency is causative of Loeys-Dietz 4 syndrome.

The patient came to our observation for investigation regarding the recent development of ocular paroxysmal events characterized by sudden downward deviation of the eyes, slight retraction of the upper eyelid, lasting several seconds and undergoing spontaneous resolution. This paroxysm occurs several times during the day and can be triggered by movements, feeding, or stimulations.

On admission to the ward, he was in good general condition. Inspection revealed no facial dysmorphism, but we noted the presence of an incisura in the tragus bilaterally and a single bilateral palmar sulcus. The anterior fontanel was normotensive and not pulsating, and the pupils were isochoric isocyclic and normoreactive to photostimulation. Deep Tendon Reflexes (DTR) were present and valid bilaterally. Neurodevelopmental abnormalities emerged upon neurological examination. Marked axial hypotonia was evident: He did not clear the airways in the prone position, and dorsal and ventral suspension maneuvers were positive. General movements were slightly variable with a prevalence of the left side and lower limbs. The grasping reflex was hypovalid, and the stepping reflex was absent. The visual engagement was present, with a semi-complete arch on the right and left. Hammersmith Infant Neurological Examination was performed and resulted in a suboptimal score.

Given the resemblance to the sunsetting eye sign, and despite the fact that our patient's downward gaze deviation was intermittent than persistent, we initially ruled out increased intracranial pressure by performing transfontanellar ultrasonography. Following this, a comprehensive ophthalmological examination was conducted, the results of which were within normal limits.

Doppler echocardiography was performed due to the elevated cardiovascular risk associated with LDS, and it revealed the presence of a small coronary-pulmonary fistula, which we deemed to have no hemodynamic significance.

There were no signs of muscle atrophy or other pathological findings.

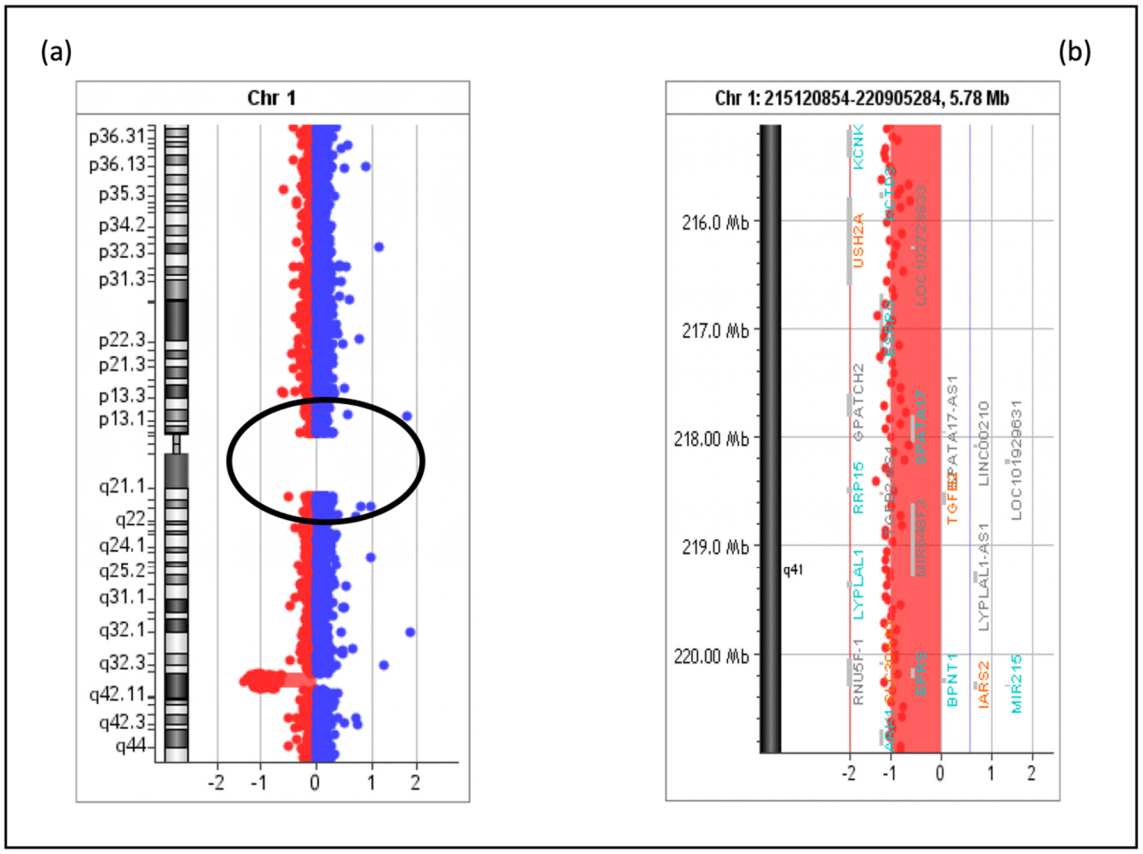

Figure 1. Array comparative genomic hybridization analysis on the DNA extracted from peripheric blood by SurePrint G3 Oligo ISCA V2.0 8x60k Array (Agilent), showing the result (a) of arr [GRCh37] 1q41 (215120854_220905284) x1 with a 5,7 Mb 1q41 interstitial deletion encompassing 17 genes RefSeq of which 6 are OMIM genes, including TGFB2, USH2A, SLC30A10, EPRS, and RAB36AP2 (b).

Given the suspicion of critical episodes, polygraphic video-EEG monitoring was conducted. The interictal video-electroencephalogram (EEG) revealed unstable, polymorphic baseline activity, with medium-voltage and theta-dominant rhythms bilaterally. The sleep-wake transition appeared normal, and physiological hypnic graph elements were identifiable. During the video-EEG monitoring, multiple episodes were observed, characterized by sudden downward gaze deviation that lasted only a few seconds and resolved spontaneously. Notably, these paroxysmal motor events did not correlate with electrographic changes.

3. Discussion

In this report, we describe the case of an infant diagnosed with LDS who presents with clinical neurological involvement and non-epileptic ocular paroxysms. To the best of our knowledge, the association between LDS and such ocular paroxysms has not been documented in the literature.

Although relatively uncommon, neurodevelopmental abnormalities such as hypotonia, delays in gross motor development, intellectual disabilities, and speech delays have been documented in the literature in association with LDS.

Fry et al. described five patients with pure 1q41 deletion encompassing TGFB2, exhibiting a neurodevelopment impairment, including hypotonia, gross motor delay, intellectual disability seizures, and migraines [20].

Gaspar et al. and Lindsay et al. described the same microdeletion in four patients with LDS type 4, three of whom had hypotonia and gross motor delay [21,22] (Table 1).

Table 1. Summary of the major studies linking 1q41 deletion encompassing TGFB2 to neurodevelopment abnormalities.

Authors	Deletions on chromosome 1q41	Main findings
Fry et al. 2022 [20]	Patient 1: 7.84 Mb deletion on chromosome 1q41(215,199,578–223,035,427) [GRCh37].	Hypotonia, gross motor delay, intellectual disability, speech delay.
	Patient 2: 1.44 Mb deletion at 1q41 (217,589,671–219,026,274) [GRCh37].	Global developmental delays, hypotonia, seizures, migraine.
	Patient 3: 3.99 Mb deletion at 1q41 (216,243,817–220,231,236) [GRCh37].	Gross motor delay, hypotonia.
	Patient 4: 2.17 Mb deletion at 1q41 (217,219,51–219,385,296) [GRCh37].	No neurodevelopment abnormalities reported.
	Patient 5: 785 kb deletion at 1q41 (218,238,773–219,024,035) [GRCh37].	Global developmental delays, ADHD and Autism.
Gaspar et al. 2017 [22]	4.74 Kb deletion on chromosome 1q41 (215,963,393-220,705,991) x1	Hypotonia with problems in motor coordination, dyslalia.
Lindsay et al. 2012 [21]	3.5 Mb deletion on chromosome 1q41 (216,672,181–220,202,575) 6.5 Mb deletion on chromosome 1q41 (215,588,712–222,145,072; GRCh37/hg19)	Mild development delay.

Note: ADHD: Attention-deficit/hyperactivity disorder.

To our knowledge, the association between LDS with 1q41 deletion and ocular paroxysmal events has never been described in the literature. Ocular paroxysmal events (OPEs) are sudden involuntary movements that involve muscles of the eyes and eyelids [23]. They are common phenomena in newborns and infants and are often related to incomplete myelination of the two major axonal pathways involved in movement genesis: The corticospinal and the corticobulbar tracts. The immaturity of these systems, in fact, does not enable efficient inhibitory control of the motor system, being able to explain the tendency of the newborn and infant to show paroxysm motor phenomena of brainstem origin [24]. Although paroxysmal ocular events are typically benign in most cases, they may be associated with a range of neurological disorders. The primary and most critical diagnostic step is the performance of video-EEG monitoring [25]. For this reason, the polygraphic video-EEG monitoring was early performed on our patient. The lack of correlation between OPEs and electrographic changes enabled us to exclude that our patient's OPEs could represent epileptic activity.

We, therefore, classified this paroxysm as a "paroxysmal tonic downward gaze", an ocular motor disorder characterized by episodic downward deviation of the eyes, lasting for seconds and occurring several times per day. In most cases, PDT is a benign phenomenon that occurs in healthy newborns in correlation with the immaturity of the extrageniculocalcarine visual pathway [26]. This resolves spontaneously within 6 months of life although it may take longer in preterm infants [26]. More rarely, PDT can be associated with neurologic and oculomotor problems and pathologies such as hydrocephalus, kernicterus, congenital stationary night blindness, gliosis, and encephalomalacia of the optic pathway and occipital cortex [23,26].

We hypothesize that, in addition to the clinical manifestations described in the literature, patients with LDS may also develop paroxysmal tonic downgaze as a distinctive feature of the disease. Moreover, connective tissue involvement, the hallmark of LDS, can lead to atrophy of the extraocular muscles, a phenomenon that has been documented in the literature as a contributing factor to other ophthalmological manifestations of the disease, such as strabismus [12,16]. This same pathological process may also play a significant role in the development of ocular paroxysms. Further investigations, such as orbital MRI, should be considered to explore this association more thoroughly.

We also hypothesize that there is a link between 1q41 deletion encompassing TGFB2, paroxysmal tonic downgaze, and neurodevelopmental phenotypes. Researchers have found that in addition to encoding proteins that are important for the integrity of the extracellular matrix, TGFB2 plays an important role as a modulator of the synthesis and metabolism of the major neurotransmitters of the central nervous system. Specifically, TGFB2 appears to be critical for the development and survival of midbrain dopaminergic neurons and hindbrain serotonergic neurons [27–29]. The neurodevelopment abnormalities could be secondary to the loss of normal function of TGFB2. Data reported in the literature suggests that hypotonia and gross motor delays are particularly frequent among patients with deletions, with 7 of 9 reported individuals exhibiting these features. While hypotonia may not be a universal feature of LDS4, its high prevalence in patients with deletions, and more broadly, in those with loss-of-function variants compared to missense variants, points to a possible genotype-phenotype correlation. Notably, deletions or loss-of-function variants appear to be more common in LDS associated with TGFB2 than with other LDS-related genes. Not all individuals with a 1q41 deletion exhibit neurodevelopmental phenotypes, indicating variable expressivity and incomplete penetrance of the associated traits. These observations underscore the potential value of a more systematic neurological evaluation in patients harboring deletions or loss-of-function variants in TGFB2.

4. Conclusions

Our patient exhibits paroxysmal tonic downward gaze, an unusual clinical feature not previously associated with LDS, as well as neurodevelopmental abnormalities, including hypotonia and developmental delays. These findings suggest that the 1q41 deletion, which is typically linked to LDS, may represent a distinct, clinically recognizable contiguous gene syndrome.

In particular, the 1q41 deletion could lead to a broader and more heterogeneous clinical phenotype than what is traditionally observed in classic LDS. While LDS is primarily characterized by connective tissue abnormalities and arterial vasculopathy, our case underscores the possibility that additional neurodevelopmental and neurological features, such as the paroxysmal tonic downward gaze observed here, may be part of the spectrum of clinical manifestations associated with this genetic alteration.

Furthermore, the involvement of TGFB2, a gene essential for extracellular matrix integrity and the modulation of neurotransmitter synthesis, could explain some of the neurodevelopmental aspects of this syndrome, as well as the atypical ocular paroxysms seen in our patient.

This case highlights the importance of considering the full range of clinical features, including neurological and ophthalmological manifestations, in the diagnosis and management of patients with 1q41 deletions. It also points to the need for further research to better understand the relationship between TGFB2 and neurodevelopmental abnormalities in the context of LDS. We recommend that clinicians remain vigilant for such atypical presentations, and that further investigations, such as genetic studies and advanced imaging, be considered in order to elucidate the full extent of the clinical spectrum associated with 1q41 deletions.

Author contributions

Raffaele Falsaperla and Piero Pavone conceived of the presented idea. Giovanni Cacciaguerra and Carla Cimino developed the theory and performed the computations. Vincenzo Sortino, Marco Andrea Nicola Saporito and Evelina Moliteo verified the analytical methods. All the authors supervised the findings of this work. All authors discussed the results and contributed to the final manuscript.

Use of AI tools declaration

The authors declare they have not used artificial intelligence (AI) tools in the creation of this article.

Ethical approval of the research and informed consent

The Authors declare that the patient's parents provided written informed consent for patient information and video to be published. Ethical approval for this study/case/case series was obtained from Policlinico San Marco INSTITUTIONAL REVIEW BOARD (APPROVAL NUMBER/2021-213571).

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Conflict of interest

Piero Pavone is an editorial board member for AIMS Medical Science and was not involved in the editorial review or the decision to publish this article. All authors declare that there are no competing interests.

References

1. Malyuk DF, Campeau N, Benson JC (2021) Loeys-Dietz syndrome: case report and review of the literature. *Radiol Case Rep* 17: 767–770. https://doi.org/10.1016/j.radcr.2021.12.024
2. Velchev JD, Van Laer L, Luyckx I, et al. (2021) Loeys-Dietz syndrome. *Adv Exp Med Biol* 1348: 251–264. https://doi.org/10.1007/978-3-030-80614-9_11
3. Schepers D, Tortora G, Morisaki H, et al. (2018) A mutation update on the LDS-associated genes TGFB2/3 and SMAD2/3. *Hum Mutat* 39: 621–634. https://doi.org/10.1002/humu.23407
4. Camerota L, Ritelli M, Wischmeijer A, et al. (2019) Genotypic categorization of Loeys-Dietz syndrome based on 24 novel families and literature data. *Genes* 10: 764. https://doi.org/10.3390/genes10100764
5. Iqbal R, Alom S, BinSaeid J, et al. (2021) Loeys-Dietz syndrome pathology and aspects of cardiovascular management: a systematic review. *Vascular* 29: 3–14. https://doi.org/10.1177/1708538120934582
6. Loeys BL, Dietz HC (2008) Loeys-Dietz Syndrome, In: Adam, M.P., Everman, D.B., Mirzaa, G.M., et al., Editors. *GeneReviews® [Internet]*, Seattle: University of Washington. (updated Sep 12, 2024)
7. Ting TW, Lai AH, Choo JT, et al. (2014) Loeys-Dietz syndrome in a Southeast Asian hospital: a case series. *Eur J Pediatr* 173: 387–391. https://doi.org/10.1007/s00431-013-2187-0
8. Loeys BL, Chen J, Neptune ER, et al. (2005) A syndrome of altered cardiovascular, craniofacial, neurocognitive and skeletal development caused by mutations in TGFBR1 or TGFBR2. *Nat Genet* 37: 275–281. https://doi.org/10.1038/ng1511
9. Baldo F, Morra L, Feresin A, et al. (2022) Neonatal presentation of Loeys-Dietz syndrome: two case reports and review of the literature. *Ital J Pediatr* 48: 85. https://doi.org/10.1186/s13052-022-01281-y
10. Erkula G, Sponseller PD, Paulsen LC, et al. (2010) Musculoskeletal findings of Loeys-Dietz syndrome. *J Bone Joint Surg Am* 92: 1876–1883. https://doi.org/10.2106/JBJS.I.01140
11. MacCarrick G, Black JH 3rd, Bowdin S, et al. (2014) Loeys-Dietz syndrome: a primer for diagnosis and management. *Genet Med* 16: 576–587. https://doi.org/10.1038/gim.2014.11
12. Wang W, Man H, Zhang J, et al. (2022) Ocular findings in two Chinese children with Loeys-Dietz syndrome. *Eur J Ophthalmol* 32: NP55–NP59. https://doi.org/10.1177/11206721211020649
13. Burgess FR, Carroll N, Young SL, et al. (2021) A short-sighted approach to high myopia-not just an eye problem. *J AAPOS* 25: 247–248. https://doi.org/10.1016/j.jaapos.2021.03.005
14. Busch C, Voitl R, Goergen B, et al. (2018) Ocular findings in Loeys-Dietz syndrome. *Br J Ophthalmol* 102: 1036–1040. https://doi.org/10.1136/bjophthalmol-2017-311254
15. Van Laer L, Dietz H, Loeys B (2014) Loeys-Dietz syndrome. *Adv Exp Med Biol* 802: 95–105. https://doi.org/10.1007/978-94-007-7893-1_7
16. Sathappan VS, Matharu KS, Coats DK, et al. (2021) Novel finding of atrophic extraocular muscles in Loeys-Dietz syndrome: a case report and review of the literature. *J AAPOS* 25: 119–121. https://doi.org/10.1016/j.jaapos.2020.11.010
17. Braverman AC, Blinder KJ, Khanna S, et al. (2020) Ectopia lentis in Loeys-Dietz syndrome type 4. *Am J Med Genet A* 182: 1957–1959. https://doi.org/10.1002/ajmg.a.61633

18. Eghrari AO, Rasooly MM, Fliotsos MJ, et al. (2020) Corneal thinning and cornea guttata in patients with mutations in TGFB2. *Can J Ophthalmol* 55: 336–341. https://doi.org/10.1016/j.jcjo.2020.03.007
19. Longmuir SQ, Winter TW, Gross JR, et al. (2014) Primary peripheral retinal nonperfusion in a family with Loeys-Dietz syndrome. *J AAPOS* 18: 288–290. https://doi.org/10.1016/j.jaapos.2013.12.013
20. Fry D, Groepper D, MacCarrick G, et al. (2022) Loeys-Dietz syndrome caused by 1q41 deletion including TGFB2 is associated with a neurodevelopmental phenotype. *Am J Med Genet A* 188: 2237–2241. https://doi.org/10.1002/ajmg.a.62758
21. Lindsay ME, Schepers D, Bolar NA, et al. (2012) Loss-of-function mutations in TGFB2 cause a syndromic presentation of thoracic aortic aneurysm. *Nat Genet* 44: 922–927. https://doi.org/10.1038/ng.2349
22. Gaspar H, Lutz B, Reicherter K, et al. (2017) 4.7 Mb deletion encompassing TGFB2 associated with features of Loeys-Dietz syndrome and osteoporosis in adulthood. *Am J Med Genet A* 173: 2289–2292. https://doi.org/10.1002/ajmg.a.38286
23. Falsaperla R, Saporito MAN, Pisani F, et al. (2021) Ocular motor paroxysmal events in neonates and infants: a review of the literature. *Pediatr Neurol* 117: 4–9. https://doi.org/10.1016/j.pediatrneurol.2020.06.001
24. Orivoli S, Facini C, Pisani F (2015) Paroxysmal nonepileptic motor phenomena in newborn. *Brain Dev* 37: 833–839. https://doi.org/10.1016/j.braindev.2015.01.002
25. Watemberg N, Tziperman B, Dabby R, et al. (2005) Adding video recording increases the diagnostic yield of routine electroencephalograms in children with frequent paroxysmal events. *Epilepsia* 46: 716–719. https://doi.org/10.1111/j.1528-1167.2005.50004.x
26. Facini C, Spagnoli C, Pisani F (2016) Epileptic and non-epileptic paroxysmal motor phenomena in newborns. *J Matern Fetal Neonatal Med* 29: 3652–3659. https://doi.org/10.3109/14767058.2016.1140735
27. Roussa E, Wiehle M, Dünker N, et al. (2006) Transforming growth factor beta is required for differentiation of mouse mesencephalic progenitors into dopaminergic neurons in vitro and in vivo: ectopic induction in dorsal mesencephalon. *Stem Cells* 24: 2120–2129. https://doi.org/10.1634/stemcells.2005-0514
28. Chleilat E, Skatulla L, Rahhal B, et al. (2018) TGF-β signaling regulates development of midbrain dopaminergic and hindbrain serotonergic neuron subgroups. *Neuroscience* 381: 124–137. https://doi.org/10.1016/j.neuroscience.2018.04.019
29. Chleilat E, Mallmann R, Spanagel R, et al. (2019) Spatiotemporal role of transforming growth factor Beta 2 in developing and mature mouse hindbrain serotonergic neurons. *Front Cell Neurosci* 13: 427. https://doi.org/10.3389/fncel.2019.00427

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